

## E C L A M P S I A

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Its association with thyroid inadequacy -  
case of pregnancy in a woman suffering  
from myxoedema.

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I n discussing eclampsia, it is well to note  
that people are not even agreed as to what ought to be  
defined as eclampsia. The reason is not far to seek.  
So far as we know there is no exact pathological ~~rea-~~<sup>cause</sup>  
~~son~~ agreed upon by everyone as the always ~~and~~ present  
determining lesion.

I shall take as my definition "The occurrence  
of epilepti-form fits during pregnancy, labour or  
puerperium, with the presence of albumen in the urine"

C a s e s are recorded where no albumen has  
been present and still classified as eclampsia. It  
is doubtful if these cases ought not to have come un-  
der a different heading, e.g. cases of epilepsy; or  
the albumen might have been so small in quantity as to  
be overlooked.

I n the first part of this thesis I shall  
deal with the pathological appearances of the differ-  
ent organs in eclampsia, referring incidentally to  
theories advanced explaining the different lesions.

U n t i l recently the kidney received most

attention as being the prime factor in eclampsia, but with more recent observations it has become evident that the kidney is only one of the many organs showing changes in this obscure disease.

Unfortunately autopsies on subjects dying from eclampsia have not been very frequently carried out in this country, but from those which have been done, and from reference to others, fairly definite appearances are noted.

#### K i d n e y.

U n t i l recently, as already stated, many, if not most obstetricians thought that this organ was primarily at fault in eclampsia. Now however the majority hold the view that it is of the nature of a toxæmia and that the kidney lesions are simply an indication of the presence of this toxæmia.

I n dealing with the kidney, we must take note of the ureters, as a theory has long held sway that pressure on the ureters by the enlarged uterus is a primary cause of eclampsia. This theory seems to be strengthened by the fact that primiparae and especially primiparae with twins, are more liable to eclampsia than ordinary cases. However these facts are capable of a different explanation. Were the mere uterine distension, and pressure on the ureters and the renal veins responsible, one would expect

eclampsia to occur in women suffering from, say, huge ovarian or fibroid tumours. Such however is not borne out by experience, and these patients do not suffer markedly from either eclampsia or uraemic convulsions.

T h e explanation, assuming the kidney to be at fault, is probably that the first confinement has really brought out the weak spot in an already damaged kidney and that presuming the cause to be a toxæmia favoured by the presence of the foetus, the mother is not capable of contending with the effete matter secreted by two foetuses. This to some extent is corroborated by the fact that if the foetus dies the mother - if an eclamptic - has a better chance - c f. Barbour's<sup>2</sup> case. That pressure has really nothing to do with the kidney inadequacy is still further borne out by a paper contributed to the British Gynaecological Journal of October 1905. In this paper Cumston<sup>23</sup> states that pressure on the ureters is no longer admitted as a cause of eclampsia. Halbertstma showed that in women dying from eclampsia, "there was often a considerable dilatation of one or both ureters". But in the light of Cumston's statement that "usually women dying during latter part of pregnancy have one or both ureters dilated, sometimes as large as the small intestine" it is difficult to trace any causal



relationship between dilation of ureter and eclampsia

I f we turn to experimental work, we find that if the ureter of the dog is constricted and a mercurial manometer placed therein, when the mercury has risen to say 50 or 60 m.m. it remains stationary.

Ludwig interpreted these results (see Starlings article p. 649 Schäfer's physiology) as determining the conclusions he had already drawn. He regards the urine as a filtrate and thought that a certain minimum difference of pressure was necessary for filtration to take place.

Heidenham contested his arguments and said the kidney has not ceased to secrete, but reabsorption is taking place. Be this as it may, suffice it to say for our purpose, that if the ureter is being constricted, then the patient is being poisoned, either because the urine is being reabsorbed, or the kidney is rendered inactive. However, had the obstruction been so great in eclampsia as to render the kidney inactive one would find Post-Mortem evidence of it in not only dilatation of the ureter, but also of the pelvis and haemorrhages into the substance of the kidney itself.

W e should expect a Hydronephrotic condition of the kidney but such is not the case oftener than in ordinary pregnancy and moreover in cases where operation has been undertaken for Hydronephrotic



kidney caused by pregnancy, although both kidneys were effected and badly effected, there was no eclampsia (see Cumston) This seems sufficiently brought out by eleven cases collected by Cumston, all suffering from Pyelonephritis of pregnancy, and not one exhibited symptoms of eclampsia.

If my contention is correct, based on Cumston's collected cases, then the kidney is not at fault in eclampsia and we must look for some new poison, and not one that is ordinarily excreted by a normal kidney. In Cumston's cases, especially in the last one, we have both kidneys thrown out of action, owing to pressure on the ureters and still no eclampsia is developed.

#### Pressure on the Renal artery and veins.

If we consider pressure on the renal artery Hermann in his studies on blood pressure showed that if the renal artery is constricted there is a diminution in urine and if still further constricted a cessation of urine. (Schäfer)

Hermann also points out if the renal vein is constricted we have, contrary to what one would expect a diminution in the flow of urine going on to total stoppage.

The explanation is perhaps difficult and Ludwig's is the most plausible that the congested interlobular veins press on the collecting tubules

and stop the secretion.

Considering our data, the clinical facts of eclampsia do not bear out the pressure theory on either artery or vein. Were such the case we should see eclampsia more frequently in ovarian tumours, where the pressure is much more severe and is often much more prolonged than in ordinary pregnancy.

<sup>25</sup>  
Herman in Albutt's system of medicine points out that no eclampsia develops either in heart or lung disease where the kidney condition is often one of extreme congestion.

I have dealt at length with the pressure theory of eclampsia, because it is still held by a good many authorities.

Dr. Boxall, in the discussion on eclampsia at the British Association 1905 says "The reason usually assigned for this (e.g. frequency of albuminuria in first cases) is that owing to the resistance of the abdominal walls the intra abdominal pressure is usually greater in first than in subsequent pregnancies. The increased pressure in the abdomen exerts its influence on the renal veins, retarding the return of blood from the kidney and setting up a process of fatty degeneration in the renal cells"

<sup>58</sup>  
Again Dr. Sikes in his article in the practitioner of April 1905 says but I think everyone must

admit that pressure on the ureters must be an exceedingly important element in the causation of eclampsia"

C o u l d increased intra abdominal pressure give rise to eclampsia, I often think of a case operated on by Professor Simpson when I was Buchanan Scholar in 1897. She was a woman sent in for operation whose history was briefly as follows.

S h e was delivered of twins one month previous to her admission. On admission she was found suffering from a very large ovarian tumour. Professor Simpson saw her and remarked on hearing that she had been delivered of twins one month previously "My good woman, how could you carry them? I hope you were given strength from a Higher Source to bear your burden". Still this lady with her huge ovarian tumour and twins had no eclampsia, neither had she albumen in her urine.

I n floating kidney where you get twisting of the ureter with Hydronephrosis (Hektoen, Vol II, Page 928) you do not have eclamptic symptoms, although there is marked congestion of the organ.

O n e would expect aneurysmal dilatation of the renal vein in pressure, especially on the left renal vein which crosses the aorta, if injurious pressure had been exercised. This condition of aorta has been noted by Senator in a case of bilateral



floating kidney. (quoted by Hektoen p. 929).

## K i d n e y.

### Macroscopical.

S i z e. This seems in cases examined to vary considerably (see Schmorl<sup>53</sup> in his Analysis of 73 autopsies)

In one of Sikes cases the kidney was Hydronephrotic, while in Bell's case, there was no enlargement of kidney and some mottling was all that could be made out.

In Ballantynes<sup>3</sup> case both kidneys were slightly enlarged. Of course we have to bear in mind that eclampsia may be superadded to an already diseased kidney and in that case we have the old condition as well.

O u t of 90 Autopsies made by Schauta<sup>56</sup>, kidney is mentioned as normal in 7, condition not mentioned in 23 - presumably not much change.

Hektoen says "the kidney of pregnancy is somewhat enlarged, pale and of a greenish yellow colour, microscopically the principal changes are found in the epithelium of the glomeruli and the convoluted tubules. They consist in cloudy swelling, fatty degeneration, and in severe cases extensive necrosis. The last is usually not diffuse but is circumscribed to irregular areas in the cortex.

Virchow has pointed out the presence of fat embolism

in the glomerular capillaries.

I t is noteworthy that Hektoen includes it in the Acute Parenchymatons or degenerative nephritis and says "Strictly speaking this should not be considered an inflammation, as the vascular phenomena peculiar to the latter are absent"

T h i s also accords with Herman's view.

Herman (in Clifford Albutt, vol.VII) says Angus MacDonald was one of the first to recognize the non-inflammatory nature of the condition. This is most interesting in the light of recent work also the description given by Professor Hamilton of Aberdeen who examined them microscopically for Angus MacDonald.

H e says "Judging from the naked eye appearances we expected that parenchymatons inflammation of the tubular epithelium in the cortex, passing into a state of fatty degeneration would be revealed.

T h e first glance at a section of the organ however showed conclusively that this was not the case, and that the lesion was not an ordinary parenchymatons inflammation".

I t consisted of degeneration of the epithelial cells of a certain proportion of the tubules in the circumferential aspect of the cortex. The degeneration appeared to be of a colloid nature. The products of these degenerated cells ran down and blocked up

more or less completely the other convoluted and straight tubules, so as to render them functionally useless, although their tissues were not diseased. Herman also quotes a case of his own where it has been described as parenchymatous nephritis by the late Dr. James Anderson on naked eye examination, but turned out when examined by Dr. Charleswood Turner as follows:- They show recent degenerative changes in the cortex - without any older or cirrhotic lesions - changes attributable to some toxic matter in the blood; granular degeneration and swelling of the epithelium of the convoluted tubes, the nuclei of which are invisible and swelling of the connective tissue throughout without infiltration of leucocytes. Some of the loops contain casts".

Herman also quotes another case examined microscopically by W. I. Fenton, presenting the same appearances.

I t is interesting to note that previous to microscopical examination being made the first two cases were considered to be parenchymatous inflammation, but after being examined microscopically the conclusion was come to that the condition was a degeneration due to some toxic matter in the blood.

O n e often sees Post-Mortem accounts of eclamptic cases, where naked eye appearances were



described as parenchymatous degeneration and presumably no microscopical examination was made.

N o w with due deference to the opinion of these, the probability is - especially after Professor Hamilton's and Herman's experiences - that had these kidneys been examined microscopically a different conclusion would have been arrived at. Such, I would venture to suggest might have been the case in Dr. Ballantyne's interesting patient - (described in Vol. XXX Ed. obstet. transactions)

I n this case the post-mortem was made by Dr. Stuart MacDonald and he described the kidney "as showing well marked parenchymatous nephritis, with intense fatty changes". There is no mention made of microscopical examination and probably, had sections been made, the condition would have turned out to be like the cases of Professor Hamilton, Dr. Herman and Cutler,

"Degeneration probably going on to fatty change"

I lay special emphasis on this, because undoubtedly the naked eye appearances of eclamptic kidneys have over and over again misled the most expert, the microscopic appearances turning out different from what they anticipated. This has undoubtedly led to the overlooking of the exact condition in many instances. Another example of the saying "More mistakes are made from want of looking than from ignorance".

<sup>6</sup>  
Berkley sums up the condition "The characters closely approach the different coagulative necroses found in infectious diseases. The lesions fall principally on the epithelial cells and vessels in contact with them" (Bouffe de St. Blaise)

M o s t observers are agreed that thromboses of capillaries and small vessels are often met with, the interstitial tissue as a rule is normal. Virchow and Winckler both found fat embolism in the Kidneys and lungs (Herman)

<sup>15</sup>  
Dienst mentions a case where there were signs of inflammation in the form of round cells in the tissues going on to fibrous formation. In some cases the kidneys appear absolutely normal on naked eye examination but microscopically as Meyer-Wirz<sup>39</sup> mentions in one case "there were no naked eye changes and albuminuria had not been detected - but microscopically there was seen degeneration and necrosis of epithelium."

#### L i v e r.

U n t i l quite recently the attention of observers was directed mostly to the kidneys, but it is becoming more and more evident that the liver suffers in the progress of this disease in fact in almost every recorded case where an autopsy was carefully made the liver has been found damaged. From the nature of the liver changes many observers have been

led to believe that the poison of eclampsia and acute yellow atrophy are one and the same.

B e that as it may the clinical phenomena in both are very much alike and the liver lesions also bear a marked similarity.

<sup>45</sup>  
Pelliet and Letienne studied this condition very carefully and found in 16 autopsies that throughout the liver was studded with small haemorrhages and also necrotic areas. Most observers since have confirmed this.

Schmorl in 71 out of 73 cases found changes in the liver. <sup>5</sup>  
Bell described a case where "on section the liver was of a dull ochre colour, the outlines of the lobules not very distinct and the liver is not enlarged."

O n microscopical examination the liver cells mostly in the central zone of the lobule show degenerative changes, and in the centre of the lobule extreme degeneration, the nuclei being faintly marked or exist as fragments only. The protoplasm has shrunk and is vacuolated.

T h i s case though clinically one of eclampsia might from microscopical examination have been one of early acute yellow atrophy. Also in two cases reported by Sikes although there were no liver changes visible to the unaided eye, on microscopical examina-



tion, the liver presented degeneration of liver cells.

I t is also interesting to note that Bell found leucin present in the urine and Sir John Williams found a similar condition present in two cases quoted by Herman. From the above mentioned facts some observers have been led to include eclampsia among the so-called "Hepatic Toxaemias", the arguments being that the liver sometimes fails to deal with the metabolic processes so active in pregnancy and then the poisonous material passes on and is dealt with by the kidneys, also to their injury.

#### L u n g s.

Herman says "These are always congested with ecchymoses underneath the Pleurae. There are often pulmonary apoplexies and sometimes blood in the bronchial tubes."

Virchow in four cases examined by him after eclampsia found fat emboli in the lungs and in the glomeruli of the kidneys, although he was quite unable to account for their origin. He concluded that there was no relation between the emboli and the eclampsia

<sup>15</sup>  
Dienst has described homogeneous masses in the peribronchial arteries, which are coloured red with alum carmine, do not stain as amyloid material, and give appearance to arteriole, as if composed of three rings, the outer being the muscular layer the middle

composed of the little masses described, and the inner clotted blood.

#### S p l e e n.

Practically all observers agree that the spleen undergoes the same pathological changes as the liver, the organ is enlarged and is the seat of numerous haemorrhages both into the substance and under the capsule. Sometimes these haemorrhages are so extensive as to almost obliterate the splenic substance.

#### H e a r t.

<sup>23</sup>  
Löhlein says that the Hypertrophy which takes place normally in pregnancy is greater here. As Herman points out this is not to be wondered at, as we get renal disease accompanying eclampsia.

In addition to hypertrophy we get numerous haemorrhages and often degeneration of the muscle cells.

<sup>15</sup>  
Dienst also described the presence both here and in the liver cells of small bodies which swell up with acetic acid.

#### B r a i n.

S m a l l haemorrhages have been described here and cortexhydrops of the ventricles. We also get oedema and flattening of the convolutions. Observers have varied very considerably in their descriptions of the brain lesions and there is nothing very definite to go upon.

## Thyroid.

<sup>58</sup>  
Sikes mentions that in one case where he examined post-mortem the thyroid of a patient suffering from eclampsia the acini were dilated but otherwise it was normal.

## Supra - renals.

In two cases quoted by Sikes in farcts are described in one supra-renal gland.

## PATHOLOGICAL CHANGES IN THE ORGANS OF THE CHILD.

<sup>20</sup>  
Fothergill made an autopsy on a child whose mother had eclampsia. The child died from compression of the thyroid gland, which was very much enlarged. Unfortunately he seems only to have recorded the changes in the thyroid and incidentally the liver.

As the thyroid was very much enlarged causing death of the foetus and had probably no relation to the eclampsia, it is sufficient to mention it here.

The Liver he found very much enlarged Lubarsch has found degeneration in the liver and also thrombi in the vessels.

<sup>52</sup>  
Schmid reports haemorrhages in the liver of a child developing fits like those of eclampsia.

## Kidney.

Many observers describe degeneration in the kidney cells and Schmid has noted blood extravasations in the kidney of the child just as in the



maternal kidney. Sikes reports fatty and granular degeneration of epithelium of tubules and enormous amount of extravasated blood.

#### B r a i n.

<sup>17</sup>  
Duhrssen mentions the case of a child dying when two days old and on post-mortem he found a huge blood clot on either side of the median line.

Albumen has also been found in the urine of children of eclamptic parents. This is not constant but it is noteworthy that Dienst and others found and described it.

Fibrin according to Kolman is increased both in the maternal and foetal blood.

F r o m the presence of so much similarity in the post-mortem changes found in maternal and foetal organs, it is no speculation to say that there is a common poison which exercises a baneful influence on both. It is well recognised that foetal mortality is excessively high in eclampsia. This is variously explained. Some think it is due to deficient oxygenation of foetal blood owing to the fits - in other words, foetal asphyxia. This may be a contributory cause but it is rational to suppose from the similarity of the eclamptic lesions in mother and child that there is an interchange of the poison - from maternal to foetal blood or from foetal to maternal blood as

the case may be.

T h a t poison circulating in the mother's  
blood can often affect the foetus, we have abundant  
proof. I have seen a case of typhoid affecting the  
mother and in consequence abortion and the bacillus  
was found in the foetus (Fordyce Ed. obstet. transac-  
tion) Syphilis variola, scarlet fever and measles may  
likewise be transmitted.

#### C a u s a t i o n o f E c l a m p s i a.

I n discussing causation of eclampsia note  
must be taken of one of the most prominent and most  
constant symptoms of this disease - albumen in the  
urine.

<sup>25</sup>  
Herman (in Clifford Albutt) gives reasons why  
eclampsia should occupy a special place from other  
kidney diseases met with apart from pregnancy. While  
acknowledging that acute and chronic nephritis, gran-  
ular kidney, lardaceous disease may co-exist with  
pregnancy, he points out that eclampsia is a disease  
per se, and that the only kidney disease - if it may  
be so called- intimately associated with it is the  
"Pregnancy Kidney"

Saft has defined this "as a disease of the  
kidney which takes rise in pregnancy alone, never  
leads to important disturbance of the general health,  
and in childbed quickly subsides".

Herman puts down the frequency of this form of disease of the kidney as occurring in about 1 % of pregnant women. We recognise its important relation to eclampsia if we consider that 1 in 5 persons suffering from pregnancy kidney develop eclampsia.

I n this form of disease we get the amount of urine either normal or diminished, and if increased it is due to some other form of kidney disease; urea is always diminished. Perhaps its chief characteristic is the rapid clearing up of the albumen in child-bed and often in 48 hours there is not a trace of albumen.

I f it persists throughout the puerperium and afterwards, the chances are that it is due to some other form of kidney disease and not the "pregnancy kidney".

T h e albumen passed with this disease is chiefly paraglobulin serum albumen rather suggesting some other form of kidney disease.

I know of no information as to the pathological appearances of the "pregnancy kidney" owing to a lack of post-mortems. Pregnant women do not die of this complaint - hence the meagreness of our information.

#### FREQUENCY AND INCIDENCE OF ECLAMPSIA.

Eclampsia is notoriously prevalent in primiparae with twins, various authorities giving different figures, but all agreeing as to its frequency in twins,



and more especially in primiparae with twins. Indeed so much is this the case that some practitioners diagnose the presence of twins in primiparae, if albumen is present in the urine.

### S t a t i s t i c s.

Herman's statistics are as follows :-

<sup>a</sup>  
Chyntreuil found that out of 683 cases of eclampsia, 522 were primiparae.

Schauta found out of 306 cases, 253 primiparae.

Schreiber found 79 % primiparae against 20 % multiparae.

O t h e r statistics vary, but all go to show the frequency of eclampsia in primiparae.

T h u s Meyer - <sup>39</sup>Wirz in Zurich gives 74.4 % amongst primiparae.

### T w i n P r e g n a n c y.

Schauta out of 341 cases of eclampsia, found 27 cases of twin pregnancy.

Hofmier, out of 104 cases of eclampsia, 9 cases of twin pregnancy. (Quoted by Herman)

Herman says, putting these cases together show in eclampsia 1 twin pregnancy to 13 normal as against 1 twin pregnancy to 80 in natural labour.

T h u s we see eclampsia is about 6 times as frequent in twin pregnancies as in ordinary labour.

Statistics as to its frequency vary consider-

ably.

33

L o h l e i n found one in 278 pregnancies.

9

B u t t n e r found one case in 600 - 25 births.

7

B o x a l l pointed out in his address on eclampsia to the British Association that for 1881 to 1903, the death rate for puerperal convulsions, is on the whole somewhat higher in Scotland than in England and Wales and in both more than double that of London, while Ireland holds an intermediate position. Also, while the death rate in England Wales and London has remained fairly stationary, there has been a considerable tendency to increase in recent years, both in Ireland and Scotland.

H e concludes " In the light of this record, the present day methods of dealing with the disease cannot be regarded with any degree of satisfaction ".

H o w e v e r it might be pointed out that although the death rate in Scotland and Ireland is higher than in London, it does not at all follow that the high death rate is due to different or indifferent treatment.

I t is well known that some places are  
remarkably free from eclampsia and the incidence  
of the disease is probably more in Scot -  
land and Ireland than in London.

I n support of this we find  
that in Wurtemberg, ( Doderlein ref :  
Harig ) there is only one case in  
3,561 , these numbers being taken from  
644,567 births.

T h i s is one possible ex -  
planation . Another is the severity  
of the cases . It is recognised by  
Practitioners who have seen a good deal  
of eclampsia , that they often get a  
great many mild , or comparatively mild  
cases and at other times a great many  
severe cases . This , I have often  
thought, accounted for the excellent Statistics  
produced by some people in one part of the  
country while in another part, apparently with  
the same treatment, the statistics were quite  
different.

I do not know whether Boxall meant  
to convey this impression or not, but it is  
certainly a possible construction and a very  
obvious one.



Frequency of Eclampsia in the same individual.

T h i s is a subject which to my mind has not received the attention it ought to have done.

W e know that there is a preponderance of eclampsia in primiparae and one would naturally ask what effect will that have on their future labours. Will it render them more liable to eclampsia or more immune?

I n the absence of definite statistics, this question is difficult to answer. Were the poison of eclampsia like that of typhoid, scarlet or small pox we would expect one attack to confer immunity or partial immunity.

O n the other hand were the cause of eclampsia due to kidney disease, we should expect an aggravated attack in each successive pregnancy.

F r o m a careful review of many recorded cases, I find very few recorded successive or frequent attacks in multiparae.

S u c h attacks however can occur and my partner Dr. Yeoman attended one case, a Mrs. T. for two attacks of apparent eclampsia; however I do not consider Mrs. T's case one of pure eclampsia as the albumen never cleared up even in the intervals when she was not pregnant. Moreover in these intervals,

the urine contained numerous granular casts and other evidence of chronic kidney trouble.

I t is interesting to note that Mrs. T. suffered from apparent eclampsia in her last two confinements and died two years after her last confinement from uremic convulsions.

T h i s case bears out what one would expect, that in kidney disease the nephritic condition becomes aggravated with each successive pregnancy and if she goes on long enough, a fatal result ensues. In regard to the second query, does one attack of eclampsia proper confer immunity from other attacks? We are bound to answer this question in the negative, although it is comparatively rare to have more than one attack in the same subject.

Sturmer (in Obstet & Gynaecolog Journal of Sept. 1904) relates a case in which a woman had three attacks of eclampsia during seven pregnancies. These occurred with the 2nd 5th and 7th confinements. He also testifies to the fact that after her 7th confinement, she left the hospital and "the microscopical and chemical examination of the urine showed that when she left the hospital after her third attack there was no disease of the kidney, and if previous disease had been present it would probably have been accentuated".

Jardine incidentally describes a case (Journal of Obstet. & Gynaecology July 1905) where he attended a woman suffering from fits. She gave him a history of fits at her previous confinement and birth of twins.

H o w e v e r as there was no albumen<sup>c</sup> he concludes it was not a case of eclampsia. Another and more illustrative case is described by Longridge<sup>34</sup> as occurring in Queen Charlotte's hospital. The history is briefly as follows. Woman aged 25, admitted in fourth month of her second pregnancy. She has had eclampsia during her first labour, the infant being still born. She had evidently no bad symptom, and urine was free from albumen, until last month, when she was sent to a home to be under supervision. On August 31st at 9.30 p.m. she was brought to hospital, when she had a fit and became unconscious. The urine was now found nearly solid on boiling. Two fits followed in rapid succession and during labour and after, she had 25 in all. Both infant and mother did well and by the sixth day the albumen<sup>c</sup> had disappeared. This is another typical case of eclampsia and taken along with Sturmers, illustrates the fact that whether one attack may or may not predispose it certainly does not confer immunity. Sturmer's case is perhaps the more instructive in that the



eclampsia appeared in 2nd 5th and 7th confinements. A priori one would have expected it to have appeared in the first labour. This however was not the case, as he attended her in her first labour, ruptured the membrane and delivered her of a live female child.

Sturmer says "I imagine three attacks in the same individual are without record".

Sturmer also says "In our experience of eclampsia two attacks occurring in the same patient are unknown. In the last ten years there have come under treatment 271 cases of eclampsia, and in no single instance among these, except this case, has eclampsia occurred twice". That Sturmer is not alone in this idea may be gathered from the fact that Dr. James Ritchie of Edinburgh the then President of Edinburgh Obstetrical Society remarked, when Dr. Nicholson described a case with three successive eclamptic attacks "I am much interested to notice that in one of Dr. Nicholson's cases the woman had three times before had eclampsia a quite exceptional condition.

Dr. Ritchie's experience - a large and varied one, evidently accords with that of Sturmer. In a subsequent article by Nicholson, <sup>43</sup> he quotes Lange's cases where out of six patients developing eclampsia they all took eclamptic fits in subsequent pregnancies.

Nicholson points out that this coincides with his own

experience in the cases he had successfully treated with thyroid extract. That Nicholson's and Lange's experience does not agree with the observations of others is clearly shown by Sturmer's cases, although it is quite possible a slight error might creep in from not having his attention specially directed to this point.

Personally I have attended 15 cases of eclampsia in the last eight years, and in only one of these has there been a definite history of more than one seizure in different pregnancies. This of course, is exclusive of the patient mentioned, who was evidently suffering from chronic nephritis. Mrs. S. Smith St. Liverpool engaged Dr. Allison whose assistant I then was, to attend her in her confinement. In her previous confinement, she had been attended by a mid-wife and nearly lost her life "owing to fits". We examined the urine at the 8th month and found a small quantity of albumen  $\frac{1}{10}$ . Immediately before her confinement it increased to  $\frac{1}{5}$ . The confinement was natural, but fits set in during the puerperium - beginning six hours and continuing for twelve hours - 16 in all. She recovered and on her urine being examined six weeks afterwards there was no trace of albumen.

S h e was again confined two years subsequently

and gave birth to twins. On this occasion although albumen appeared in the urine as before, she had no fits at her confinement but had periods of unconsciousness or coma. These came on  $2\frac{1}{2}$  hours after her confinement and lasted for two days.

A s on the former occasions the albumen cleared up and she made a complete recovery.

T h e s e cases serve to illustrate the fact that although eclampsia in subsequent confinements may be rare, still it is liable to occur. They would also serve to negative the infectious theory of Stroganoff as they conclusively prove that no immunity is conferred. That certain cases of repeated eclampsia in the multiparae may have been overlooked is not to be wondered at when we consider (1) The death rate is fairly high, about 20%; this deprives us of 20% of material for future observation (2) Cases in hospital are very often lost sight of, and may be attended to elsewhere if subsequent attacks develop. (3)

Eclampsia being a comparatively rare disease (1 in 500) most practitioners have few opportunities of watching successive pregnancies. (4) A certain number of cases refuse to become pregnant a second time fearing the consequence. I know of one such case I attended five years ago in eclampsia. Making due allowance for this, statistics do not bear out



Nicholson's and Lange's views that successive fits of eclampsia are the rule e.g. in Lange's cases in 100%.

W e r e this the case, then we should have a great preponderance of eclampsia in multiparae.

Taking statistics quoted by Herman that eclampsia occurs in about 80% primiparae as against 20% multiparae, and taking death rate at about 20% in primiparae, although it is lower than in multiparae, we should have at least 64% of multiparae according to Nicholson and Lange, not only liable, but almost certain to develop eclampsia in their subsequent confinements.

Assuming that only 50% of the 64% afterwards became pregnant and adding the 20% arising de novo in multiparae we should have at least 74% in multiparae and the proportion would be reversed.

S u c h is not the case and while admitting that cases may and do occur, we are forced to the conclusion that Nicholson's and Lange's observations were not sufficiently numerous to warrant their conclusions.

#### T h y r o i d   I n a d e q u a c y .

M y attention was first called to this by  
43  
Nicholson's original paper read before the Edinburgh Obstet. Society and published in their transactions

(Vol 26 - 184) Ever since I have used thyroid in eclampsia when a favourable opportunity offered with what results I shall detail under treatment. In this and subsequent papers Nicholson adduces very strong arguments in support of his contention. I shall now describe a case I attended  $3\frac{1}{2}$  years ago, and then discuss Nicholson's views.

P R E G N A N C Y I N A W O M A N  
SUFFERING FROM MYXOEDEMA.

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T h e few recorded examples of this condition,  
and the interest attached to it in the light of recent Investigation especially by Nicholson serve as an excuse for incorporating it in a Thesis on Eclampsia.

Shaw in his recently published work on Organs. Therapy page 24 states that in Myxoedema, the genital Organs show impaired development, hence the rarity of pregnancy in this condition.

H e <sup>26</sup> quotes Hergotts case of pregnancy in a Crepin 18 years of age. This case has frequently been referred to by Nicholson, as she developed Eclampsia during her confinement.

T h e only other case I know of is described by Ord <sup>44</sup> in Clifford Albutts system. She bore two

children after myxoedema was well established. This case was carefully investigated by Ord <sup>thirty</sup> ~~thereby~~ nine years ago, and I shall have occasion later to refer to his observations.

T h e case described in this Thesis adds yet another to the number in this rare condition.

F o r the history of the case up to the year 1900, when I first attended her I am indebted to my partner Dr. John Osborne Blunden. He attended her practically from infancy, and knew her before she developed Myxoedema.

S h e had three children, in all of which confinements Dr. Blunden attended her, and the last of these took place 17 years ago, when Mrs. L. was a strong robust woman- 30 years of age.

H e r confinements were quite natural, with no post-partum haemorrhage or other complication. Three years after the last of these confinements, or 14 years ago, Dr. Blunden was called in because "Mrs. L. had gone peculiar".

H e found her hands swollen, also her face, and to a less degree her ankles. Her mother - who is still alive - informed him that her memory was also affected and that she appeared forgetful.

W h e n asked a question she was slow to answer, but once started she would go on indefinitely.



in fact Dr. Blunden described her as garrulous.

Dr. Blunden thinking she was suffering from Chronic Bright had specimen after specimen of urine examined but no trace of albumen could be found, neither did the pulse suggest Chronic Bright, as there was no high tension.

She was given a largely milk diet, put on iron and treated as if suffering from Brights disease.

This treatment continued for 12 months with the result that she became steadily worse; the swelling of the hands and face gradually increased, her hair lost its natural gloss, and although only 33 years of age she was partially bald; her skin felt harsh and dry; and her teeth fell out one by one; the mental condition remained much about the same, as far as he could judge.

It then occurred to Dr. Blunden, and his partner the late Dr. C. Yeoman, that the condition was Myxoedema. They accordingly administered sheeps thyroid obtained from the local butcher, and after six weeks treatment, the effect was very striking. The swelling of the face and hands quickly although never absolutely disappeared, and the general physical condition improved greatly.

The mental condition improved much more

slowly, but as time went on, and with continued thyroid treatment her memory improved, as also did her mental condition. She was then put on thyroid tabloids (5 grs) <sup>in each</sup> at first taking two a day, until she regained her normal or nearly normal condition and afterwards taking one a day.

S u c h was her condition, when I first attended her 6 years ago. She was taking her thyroid almost regularly one tabloid per day, and although under treatment for so long, no one who had ever seen a case of Myxoedema could possibly mistake it.

H e r memory had now improved, and although not bright mentally, she could not be called stupid; When going about, her movements were very slow and she walked with deliberation, as if carrying a burden. Her speech corresponded with her gait, and was slow and laboured contrasting with Dr. Blunden's description, in fact her whole demeanour could only be expressed as Lethargic - She had still distinct fullness of the eyelids, alae nasi, and a good deal of swelling over both <sup>v</sup>clapicles. Her hands were also characteristic; as was also the dry condition of the skin; the toothless mouth and the partially bald head; In 1901, her husband, a policeman, died and being in rather straightened circumstances <sup>she</sup> did not attend to her thyroid treatment, as well as she

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1902.





might have otherwise done. Photograph No. 1 taken by a local Photographer in 1902, gives a good impression of how she looked, when she became pregnant. At this time she was keeping house for a batchelor of intemperate habits with the result that she became pregnant. The stoppage of the menses, which had previously been excessive did not excite her suspicion and at first she herself attributed it to the menopause - being now 44 years of age. She accordingly said nothing about her condition for 4 months and went on taking her tabloids as usual. At the end of 4 months she consulted me about the menses and on examination I found she was pregnant. I gave her sufficient thyroid to last her for one month at the rate of 4. (5grain tabloids) weekly, which she had now been reduced to, and she went away. After this, I didn't see her for 3 months, and attributed it to the fact that she was ashamed of her condition.

Meeting her at the end of 3 months, I enquired where she got her thyroid from, and she said "I have had none for two months and feel better than I have done for a long time". She looked much better but still retained the "Myxoedemic Appearance". She asked me if she ought to resume taking the tabloids, but I told her it was not necessary, as I concluded she had come to an age when she could dispense with



them. However I asked her to send a specimen of urine to the Surgery, and let me know if she suffered from headache giddiness or if any of her old symptoms returned. The urine on examination showed S.G.1019 and not a trace of albumen or sugar.

Satisfied on this point: as Eclampsia was the only complication I feared, I called and warned her mother to let me know if at any time her daughter was taken ill. The old woman said that her daughter's health had been a great deal better for the past 2 months, in fact, she said, she was better than she had been for years.

I saw her from time to time, until her confinement came off, <sup>which</sup> ~~this~~ turned out to be quite a natural labour, and although she was fairly old, and had had no child for 14 years previously the labour only lasted 3 hours. After the child was born, while waiting for uterine contraction, I noticed she was bleeding very freely. Accordingly I expressed the placenta by Credé's <sup>E'</sup> method and instructed the Midwife to get me some hot water. The haemorrhage continued not with standing pressure on the uterus, and hot water douching, for about 15 minutes. Ultimately by putting the child to the breast, I got the uterus to contract sufficiently and the haemorrhage stopped.

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L

T h e quantity of blood lost had no evil affect on her, the child was a well nourished female child, and altogether the confinement was most satisfactory. In 14 days she was up and going about her duties as usual. Exactly 1 month after her confinement she called for some thyroid, as the swelling of the hands and face was returning. She complained chiefly of her hands as she said they felt so stiff and useless. Her speech if anything was slower and more laboured and her movements seemed even slower than usual. I gave her some tabloids of compressed thyroid (5 grs in each) and instructed her to take 2 daily, and as she felt better she could reduce them to one daily.

T h e child was bottle fed, as she had not a sufficiency of milk and died when 10 months old from infantile diarrhoea. I do not think the Myxoedema of the mother was at all a factor in the child's death, as a good many other children succumbed to the same complaint at this time.

T h e other photographs, which I had taken 3 years after this confinement show her general appearance in March 1906. The raised position of the eyebrows described by Ord and attributed by him to the effort of keeping the eyelids apart is well shown in photograph No. 2. also the oblique direction of the

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4



5





eyelids (The Mongolian feature) to a certain extent can be detected. This photograph also shows the heavy though not obliterated features, and the fullness in the supra-cl<sup>u</sup>avicular regions.

T h e spade hands described by Gull are also well shown in this and the other photographs.

Another point which can be seen in the photographs is the skin condition, especially of the hands. Photograph No. 3 shows as it were a super-abundance of skin on the hand, so that the normal folds of the skin are greatly exaggerated. This is no doubt due to thickening of the skin and also absorption of the Myxoedematous element by the thyroid treatment which she had undergone. On pinching up a fold of skin between finger and thumb it felt harsh and dry, almost as if composed of scales. This latter dryness is due to lack of perspiration, which never seemed to have been re-established. Photographs 4 5 and 6 show the scanty condition of the hair on the front part of the Scalp. Even with thyroid treatment the hair never recovered its natural gloss. In the axillae a certain quantity still remained but on the pubis it had quite disappeared.

I n discussing this case several points struck me as both interesting and peculiar.

(1) The long period of sterility between 3rd and 4th child - 14 years. This is the general experience for as Shaw points out there is a degeneration of the reproductive organs in this disease.

(2) The fact that for at least the last four months of her pregnancy she took no thyroid whatsoever and felt better than when taking it.

(3) The fact that during pregnancy, confinement and puerperium. She had no suspicion of either Eclamptic or pre-eclamptic symptoms.

(4) Post-partum haemorrhage which was never alarming but most significant.

<sup>44</sup>  
In Ord's case described in Clifford Albutts system of Medicine, we find that exactly the same thing happened. Her confinements previous to the establishment of the disease were perfectly normal, but in the two confinements after the disease was established she had severe post-partum haemorrhage.

Ord's case further agrees with my experience in that she had no suspicion of Eclampsia either before during or after Labour.

2nd Point.

In regard to the second point that she took no thyroid for the last four months of pregnancy, Ord's



observations would seem to confirm this. He states "In connection with Pregnancy in Eclampsia, fluctuations in the swelling of the body often occur. There is sometimes an increase more commonly a decrease so that in early stages (of the disease) the patient may resume almost a natural appearance during pregnancy".

These deductions he probably drew from his own case, and although he attempts no explanation, his observations are most interesting. In my case she left off taking thyroid at the end of the fifth month, but whether she could have done without it at an earlier stage in her pregnancy, I am not in a position to state. Had my attention been called to Ord's statement before seeing her, I might possibly have withheld thyroid as soon as I knew she was pregnant.

S e v e r a l possible explanations of this phenomenon suggest themselves, and I shall discuss them in detail.

- (1) Mrs. L's thyroid gland was simply inadequate previous to becoming pregnant and the so-called physiological hypertrophy took place with the result that she was able to go through her confinement almost normally.

It is a well known fact that the thyroid may be inadequate or partially inadequate and to these latter cases Reverdin applied the name Myxoedema "Frustre". This

however applies more to cases where only a few features of Myxoedema are present.

D r. Russell of Edinburgh describes what he considered to be a typical case of Myxoedema, and after treating her with thyroid gland most successfully found she could afterwards go on without it.

M r s. L's case however does not come under either of these catagories. Hers was not a case of Myxoedema "frustre" neither was it a similar case to Dr. Russells.

E v e n supposing the stimulus of pregnancy restored the gland function. We should at least expect it to go on for some considerable time after labour. This argument would also apply, if we suppose that either the parathyroid or p<sup>r</sup>o<sup>r</sup>uctary took on the action of the thyroid.

S u c h however was not the case for we find that shortly after labour was terminated Mrs. L. had again to resort to thyroid treatment, and has

gone on with it ever since.

*Continued* ~~Substituted~~ ~~substitutes~~ The fact that Mrs. L. so long on thyroid treatment would point to her gland being almost in abeyance, and as post-mortems on bad cases of Myxoedema often show the glandular substance replaced by fibrous tissue, it is hardly possible if her gland were in this condition, to suppose that pregnancy or anything else could affect it.

In regard to para-  
thyroid and Hypophysis Hektoen  
states ( P. 503 )

" The extent to which a compensation for the suspension of the function of the thyroid in man may be furnished by the parathyroids and hypophysis has not yet been established."



C a n FOETAL THYROID supplement the  
MATERNAL.

T h i s was really the explanation that first occurred to me, and while one would almost imagine that the foetal thyroid would find sufficient work in stimulating its own metabolism, still it is worthy of consideration.

<sup>3</sup>  
Ballantyne incidentally pointed out that it was impossible for the foetal thyroid to supplement the maternal, as far as Iodo-thyr<sup>in</sup>m is concerned, because the foetal structure contains no Iodine. On looking still further into this matter, I find that <sup>40</sup>Merva and Stoltzner found no Iodine in the thyroid of young infants.

T h i s at first seemed conclusive but it occurred to me "Is Iodine essential for the activity of the thyroid gland ?

Robert Hutchison states "Although the active principle of the thyroid in the adult contains Iodine, there is no proof that the presence of Iodine is essential for its activity" Moreover Lang (quoted by Shaw) has advocated the giving of "Aiodin a substance which only <sup>Contains</sup> .4 to .5 % of Iodine. This was introduced because several observers thought that the therapeutic effect of the gland did not depend on Iodine alone.



The spleen contains relatively more Iodine than the thyroid and still has no effect on a-thyrea (Shaw)

P e r h a p s the strongest argument against Iodine being essential for the activity of the thyroid gland <sup>is</sup> to the observation of Gauthier <sup>22</sup> "That the thyroids of cattle contain not a trace of Iodine, and yet they are capable of producing as much therapeutic effect, as the glands of sheep, which contain a relatively large proportion of Iodine." A still further argument against Iodine being essential is the varying amount of Iodine in the gland at different times, under different conditions.

I have adduced sufficient evidence to show that Iodine is not yet proven to be essential for the activity of the thyroid.

N o w let us see, if there is any evidence of the activity of the foetal thyroid and its power to supplement the maternal. From the nature of things such evidence must be purely circumstantial.

I n infancy the thyroid weighs 0.16 % of the body weight, while in the adult it weighs only 0.5 % (Hektoen) or according to Krause in infancy the proportion is 1 to 240 or 400 (Quoted by Shaw)

In the adult 1 to 1800

T h i s in itself would lend weight to the idea that the thyroid is most active in infancy and

theoretically is less active as we advance in life. Curiously enough the latter part of this statement is borne out by facts, as Kocher found on excising human thyroids. He observed that the younger the subject, the more marked the evil after effects, and that little or no evil effects may follow excision in elderly people.

We might now ask - is interchange between foetal blood and maternal blood likely to take place? We know that during foetal metabolism effete products must be got rid of by the maternal blood and that the maternal blood must have a two-fold function

(1) Aeration and nourishment of foetal blood

(2) Getting rid of effete products from foetal blood.

Moreover we have the experiments of Baron and Castaigne, who found that if they injected toxins into the foetal blood, they poisoned the Mother; also that transmission of the injected toxin ceases on the death of the foetus.

In the light of these experiments it does not seem so far fetched to suppose that given an active foetal thyroid, and an inadequate or suppressed maternal, the foetal blood may have the power of transferring more than effete matter to the maternal blood. The clinical facts observed in Mrs. L's case

would certainly seem to support this view. The fact that almost immediately after the labour was over - one month - she had again to resort to thyroid treatment would point either to the presence of the foetus or placenta in utero as being the cause of her improved state during pregnancy.

### 3 r d P o i n t

Mrs. L. showed neither pre-eclamptic nor eclamptic symptoms during pregnancy labour or puerperium.

I f the occurrence of Eclampsia in Hergott's case was anything more than a coincidence and was due to the thyroid condition, then it is certain that there is some vital difference between congenital myxoedema and acquired myxoedema.

A s I shall have occasion to point out later the pathological condition of the thyroids in Cretins and in Myxoedemics, as shown by Post Mortem examination, is very much alike, and if Cretins develop Eclampsia so also should Myxoedemics.

H o w e v e r , both Ord's case and my own prove conclusively that this is not so. Had such been the case, we had practically every supposed predisposing circumstance present in Mrs. L's case. She was a Myxoedemic; had given up taking thyroid extract; was subjected to great nervous worry on account of her



condition - the child being illegitimate - and still she had no symptom of Eclampsia.

#### 4 t h P o i n t

T h e Post-Partum haemorrhage, I shall refer to in discussing Nicholson's views.

I shall briefly epitomise Nicholson's views as contained in (vols: 26, 27 28 and 29) Edinburgh Obstetrical transactions, and other Journals, and then discuss them seriatim. Nicholson's thoughts were first directed to the probability of thyroid inadequacy being a potent factor in Eclampsia by <sup>a</sup> Lange's observations - These and his other reasons for advocating his views I shall endeavour to state briefly in this Thesis

#### NICHOLSON'S OBSERVATIONS ON THYROID.

1. Lange noted twenty five pregnancies where physiological hypertrophy did not occur; in 20 of these there was albumenuria.
2. Lange found that thyroidin diminished the physiological hypertrophy of pregnancy
3. Murray found that in some cases thyroidin diminished the hypertrophy in goitre.
4. Lange found that thyroidin has a diuretic effect in albumen<sup>i</sup>uria of pregnancy, although not largely diminishing the albumen<sup>i</sup>.

Nicholson further noted the similarity of

the oedema in eclampsia to that in myxoedema. He also suggested that the eclamptic symptoms were due to spasm of the renal arterioles (Albutt's view) brought about by a toxin in the blood and compares this with <sup>a</sup>leach poisoning. He found from pulse tracings that arterial tension was greatly increased towards end of pregnancy in eclamptics with corresponding diminution of urine; and other symptoms, attributed by him to toxoemia, were headache, diarrhoea and eye conditions

T h i s spasm in eclampsia - he suggests - may be due to the presence of toxins in the body impairing the function of the thyroid, and leaving the <sup>v</sup>saso-constrictor action of the supra-renals unopposed: it may also be due to inadequacy of thyroid itself, and the same result takes place.

H e further points out the effect of thyroid inadequacy upon urea secretion, showing how it may be diminished. He further hypothecates from comparing experiments of Bradford (quoted by Schafer) on the Kidney that the same thing happens - wasting of muscles - as when thyroid is administered as an anti-fat.

H e surmises that thyroid exercises some control over the kidney substance and if part of the latter is removed, it exercises its superfluous energy in metabolism.

H e explains the liver condition by supposing

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that the thyroid being in obedience, the liver is called upon to do extraordinary work - hence it strikes.

Nicholson further explains the benefit of morphia in eclampsia on similar grounds, as thyroid extract. He, in fact, supposes that it lessens the arterial spasm, due on his hypothesis either to lack of thyroid secretion or impairment of its functions by some toxin. He also explains the use of saline infusions as advocated by Jardine in the same way.

In (Vol 28 Edin. Obstet. Tran) Nicholson deals with the auto-intoxication of pregnancy, pointing out its liability to occur in this condition. He says "The functions of the thyroid gland are largely concerned in defending the organism against the action of toxin which are elaborated during the process of metabolism"

In supporting this he quotes Lange's cases, where in 22 in which physiological hypertrophy did not occur, 16 had both albuminuria and tube casts, and in six eclampsia developed. This condition he attributes to toxin circulating in the blood and not combated by thyroid secretion.

He further quotes Hergott's case of a cretin developing eclampsia, and the experiments of Verstroeten and Vanderlinden on cats.

These observers found that on partial excision of the thyroid of cats, these animals when



pregnant, developed eclampsia.

L a t e r he sums up reasons for giving thyroid in eclampsia, as follows "A substance which so powerfully stimulates both metabolism and elimination; increases largely the secretion of urine, and the excretion of urea and other urinary bodies; acts as a specific vaso-dilator thus leading to perspiration and lowering of the blood pressure; cannot fail to prove a potent means of safe guarding the pregnant woman from the effects of toxic poisoning"

A l s o in exhibition to eclamptics or pre-eclamptics he points out that nearly always it increases the solid constituents of the urine, e. g. urea, and postulates that "Thyroid seems to possess a definite action in favouring the elimination of the solid constituents of the urine, and in this way certain poisonous products of metabolism are passed out of the blood"

H e further goes on to say that it favours the production of urea, which in itself is a most powerful stimulant to urinary secretion.

I n a further communication (Vol 29 Edin. Ob. Tran) he explains his reasons for giving thyroid and states that in pre-eclamptic symptoms, he gives it to re-adjust the processes of metabolism in such a manner as to complete more fully the metabolism of nitrogenous

substances. In later stages he gives it in order to get the Kidneys to secrete again.

M o s t of Nicholson's views could be explained either without reference to thyroid inadequacy or on a totally different supposition. Lange's observations on the normal hypertrophy of the thyroid gland in pregnancy - if correct - are significant, but they lack confirmation. Personally, I have made repeated observations on a number of pregnant women and have not been able to satisfy myself of any physiological hypertrophy.

T h a t hypertrophy does occur in a certain proportion of pregnant women has been recognised for a very long time; that it occurs as a physiological process in every pregnant woman is more questionable.

<sup>46</sup>  
Playfair in his Midwifery text book states "From the earliest ages authors have thought that the occurrence of conception might be ascertained by certain obscure signs, such as peculiar appearance of the eyes, swelling of the neck etc etc., all of them are far too uncertain to be of any value. Personally I am inclined to agree with Ballantyne <sup>3</sup> "From his own observations lately, he believed it was exceedingly difficult to say whether the thyroid was enlarged or not. They were almost entirely dependent on the isthmus of the thyroid for their estimation of size, unless the gland was much

enlarged or greatly diminished"

A n o t h e r fact that might tend to be very misleading is one Professor Simpson always insisted on in his lectures. He said the pregnant woman never puts on flesh; as a rule she becomes thinner. The mere fact of losing fat would tend to make the thyroid more prominent.

T h a t hypertrophy does occur in a certain proportion of pregnant women, Lawson Tait drew attention to over thirty years ago. Tait had seen twenty cases in Birmingham and gave short notes of a dozen. That he regarded these as normal is not the case, but as abnormalities, for he points out that all these women were subject to severe haemorrhage, not only during labour, but during ordinary menstruation (Quoted by Underhill, Ed: Obstet: Transacts Vol 26) This observation of Tait's is of peculiar significance and to my mind the thyroid hypertrophy during pregnancy is capable of a very different interpretation than a purely physiological one. When it does occur it is more probably a congestive hypertrophy and not a purely physiological one.

Ord, in his article in Albutt's system of medicine (Vol. 4 Page 474) points out that myxoedema is pre-eminently a disease of married women. He attributes this to the fact that congestive changes



occur during menstruation and pregnancy, and that these are of a destructive nature leading afterwards to atrophy of the glands. He says "There can be no doubt of the frequent existence of an active congestion bordering on an inflammatory condition, occurring in the thyroid gland during menstruation. Such changes are distinctly marked in exophthalmic goitre. It appears to me that probably the atrophy of the gland productive of myxoedema is frequently due to inflammatory destruction of the gland tissue" If Ord's theory is correct then a hypertrophied thyroid during a pregnancy would be much more akin to a myxoedemic thyroid than a physiologically enlarged one. Lawson Tait's cases more than bear this out. He states that all these cases had severe haemorrhage during labour, and at their menstrual periods.

N o w this is exactly the condition of the myxoedemic woman. In my case of Mrs. L. while not under the influence of thyroid, she had excessive menses, and the only complication of labour was a rather severe haemorrhage. Ord's case, where the woman bore two children after establishment of the disease, is another striking example of the same thing. She had no complication except severe post-part<sup>u</sup>m haemorrhage on both occasions while her other confinements were normal. This, to my mind, might be

considered as pathognomonic of Thyroid inadequacy during pregnancy - I mean post-partum haemorrhage.

T h e tendency to bleeding in myxoedemics is well known, and such a trivial thing (Ord) as the extraction of a tooth which has been hanging loose, may lead to haemorrhage so intractable that it goes on for days.

O n comparing the haemorrhagic condition of myxoedema with eclampsia, we find that nearly all authorities remark on the small amount of post-partum haemorrhage in the latter.

<sup>27</sup>  
Dr. Jardine of Glasgow, who has had a large experience of eclampsia says, in relation to <sup>occurrence</sup> ~~a case~~ of post-partum haemorrhage in a patient suffering from eclampsia "In my experience of eclampsia which has been a fairly large one, I have only seen one other case of post-partum haemorrhage although many of them have been under chloroform for a very long time and the uterus has been purposely allowed to relax" With this view Dunlop entirely agrees (See Article B.M. Journal Decr 23. 1905). That the giving of thyroid reduces the hypertrophy of the gland in pregnancy is no evidence of its being physiologically enlarged. Murray (Edin. Med. J. Aug. 1900) has used it to reduce the enlargement of goitre and has had some measure of success. This latter is undoubtedly pathological

and the same might apply to the former.

### HIGH ARTERIAL PRESSURE

D o e s it exist and if so is it harmful ?

Nicholson lays great stress on high arterial pressure and the part it plays in eclampsia. Herman (in Albutt) says there is not a particle of evidence to show it is a bad thing to have high arterial pressure -assuming of course it is present. Ballantyne has studied it in three cases and finds that even if the pulse be small pressure is high before a fit and it rises enormously during a fit to fall again in the interval

<sup>30</sup>  
T h i s partly agrees with Kroenig (Verhand  
d. deutsch Gesell J. Gyn 1901) who found that contractions of the uterus and the fit caused the blood pressure to rise enormously. <sup>55</sup> Schroeder investigated many cases to find if he could foretell a fit by a rise in blood pressure but was not successful.

H e r e again we have no evidence that in myxoedema there is a great rise in blood pressure, which we should naturally anticipate if Nicholson's theory were correct.

Ord states that there is no such change in arteries in myxoedema as would indicate chronic Bright and as high tension is usually associated with chronic Bright I presume he includes this. Certainly in the case I have under observation there is no high tension,



the pulse being steady, regular and easily compressible, quickening after administration of the extract

#### EXCRETION of UREA

T h a t the giving of thyroid increases the amount of urine and also of urea excreted in Eclampsia is generally though not universally admitted. This however only proves that it is a useful drug in the treatment of the condition, and we possess other remedies which although apparently having a very different action, produce the same result. Thus morphia when given in heroic doses often produces diuresis, and at the same time increases the total quantity of urea; in fact any remedy establishing diuresis in Eclampsia, also seems to increase the amount of urea.

I t is quite true that thyroid does the same though to perhaps a less extent when given in Myxoedema, but I would venture to give a different explanation of the cause. In Myxoedema we have suppressed metabolism due to lack of thyroid secretion, and as soon as it is supplied metabolism is again established and as a natural consequence urea appears - increased output of urea in myxoedema is simply an indication of renewed metabolism. In Eclampsia we have no reason to suppose that metabolism is in abeyance, and the good results obtained from the treatment by morphia would seem to contra-indicate this. Morphia probably acts as

Nicholson suggests - by relieving spasm of the arter-  
<sup>0</sup>ies and thus re-establishing the secretion of urine

<sup>in</sup> It more than probably has a further action in  
exhibiting metabolism, and giving the Kidneys a chance  
to get rid of the already accumulated urea, or its  
precursors.

I do not know that morphia has been advocated  
in the treatment of myxoedema, but from what we know  
of its action, I should fancy it would suppress rather  
than stimulate metabolism, with <sup>71</sup>careful results.

A n o t h e r point is the fact that in the  
administration of thyroid, one has to produce "Thy-  
roidism" before the excretion of urea is markedly in-  
creased. Thus we get the "pathological" and not the  
"physiological" effect of the substance, which seems  
to me to be against Nicholson's view.

#### BRADFORDS EXPERIMENTS.

T h e s e as Schaffer points out are very dif-  
ficult to explain. Willgerodt <sup>67</sup>produced uraemia by  
conducting both ureters into the peritoneal <sup>71</sup>cavity  
and concluded that uraemia was caused by some sub-  
stance secreted by the Kidneys themselves.

<sup>e</sup> Ajillo and Parascandolo experimented with the  
view of showing that uraemia was due to an internal  
secretion furnished by the Kidney.

W h i l e it is quite possible that uraemia

and also Eclampsia may be due to this, their experiments are not conclusive. Were they capable of proof then Bradfords experiments might be explained on the same or a similar hypothesis, and the condition noted by him on excision of large portions of kidney substance might be analagous to pancreatic diab<sup>o</sup>etes. Here we have either got a perverted secretion or lack of secretion, and consequently great and rapid tissue waste.

W e might also assume - according to these observers - that the Kidneys secreted a ferment which acted on the solid constituents of the urine in such a way that only the poisonous ones found their way into the urine and those rendered innocuous by such action were retained. Remove the Kidney secreting substance, and we have those substances retained which rapidly break down the tissues. This of course is a purely hypothetical explanation of a very puzzling fact.

#### A N T I T O X I C P O W E R o f T H Y - R O I D .

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Nicholson says that the thyroid has the power of defending the organism against toxins, and hence its benefit in Eclampsia.

T h i s subject has been investigated by



<sup>64</sup> Torri. He found hypersecretion of <sup>0</sup> colloidal in infectious diseases; that <sup>0</sup> colloidal material has the power of destroying organisms, further that in acute and chronic infections there is epithelial proliferation and new formation of thyroid tissue

<sup>28</sup> Kashwamura, working on similar lines was unable to confirm this

<sup>48</sup> Remedi considers that the colloidal substance of the gland neutralises ~~toxic~~ substances, since no necrosis of glandular structures takes place after their injection, neither do we find infiltration of leucocytes, as we should do were the toxins not neutralised.

It is however noteworthy that the substances separated from the gland have not borne out these observations. Thus for instance the Thyro-~~an~~<sup>t</sup>~~pitoxin~~<sup>x</sup> of Fraenkel has no proven therapeutical value.

#### PARATHYROID INADEQUACY A CAUSE OF ECLAMPSIA

True myxoedemics and Cretins are supposed by some to have suppression of thyroids with active parathyroids

The former (as expressed by Shaw page 20) secretes a substance which subserves various metabolic changes taking place in the body; in the absence of the thyroids the morphological changes taking place in

myxoedema and Cretins are the chief features noticed, as if the thyroid secretion acted as a stimulus to the various metabolic processes occurring in the tegumentary and other tissues; the parathyroids on the other hand secrete internally a substance which appear to neutralise various products of the metabolism initiated by the thyroids; in the absence of the parathyroids these toxic substances produce nervous phenomena tremors tetany psychical disturbances"

This is the theory propounded by Vassale and Generali and is supported by considerable experimental evidence

<sup>36</sup>  
Thus Lusena found that if an animal developed symptoms of tetany after removal of the parathyroids the subsequent removal of the thyroids led to the alleviation of the tetany.

On the strength of this theory the myxoedemic symptoms e.g. swelling &c., are attributed to absence of the thyroid and the nervous phenomena to parathyroid inadequacy.

Unfortunately for this theory in its bearing on Eclampsia, and with special reference to Hergotts Cretin, the only reported case of examination of the parathyroids in a Cretin, (Reported by Maresch and quoted by Shaw) showed the parathyroids normal with atrophy of the thyroid.

Now one would expect no eclamptic symptoms

if the parathyroids were normal and the less so if the thyroid were atrophied.

Nicholson also lays great stress on the myxoedemic character of the oedema in Eclampsia pointing to suppression of the thyroid function. Now were the parathyroids partially normal, we should not expect spasms in these cases, as they have less metabolic toxins to neutralise, owing to suppression of the thyroid. Moreover we should not expect so much benefit to accrue from thyroid treatment, as we would be adding fuel to the fire, and the rational method would be to administer parathyroid.

A p a r t from this we have the fact that persons dying from myxoedema often develop tetany. Here we have undoubtedly suppression of the thyroid so that the parathyroid even if partially suppressed should have nothing to do.

41  
Moussn found that after removing thyroids and parathyroids from animals, the symptoms of tetany - produced as he thought by thyroid stimulated metabolic products in the blood - could be neutralised by watery solution of parathyroid secretion. Here I may remark that in all excisions the gland is suddenly removed and hence a great strain is thrown on the nervous system. This sudden removal of an all important gland may of itself produce tetany; in Cretinism and myxoedema the



withdrawal is more gradual thus enabling the nervous system to become more accustomed to the change.

I t would be interesting to know if Moussn injected watery extract of thyroid into his already thyroidectomised animals and found the tetany made worse. Such would be the inevitable result if his conclusions are correct.

T h e beneficial results of thyroid extract in Eclampsia are attributed by those holding the view of parathyroid inadequacy to the fact that sheeps thyroid generally includes the internal parathyroid.

I t is worthy of note that when parathyroid has been given separately except in one case described by Moussn of exophthalmos, its therapeutic advantages are practically nil. <sup>38</sup> Mac Cullum has also administered it in exophthalmos, but so far with no effect, and considerable doubt is cast upon its curative effect even in Moussn's case.

I n discussing Nicholsons views I have tried as far as possible to base my evidence on facts ascertained, and while admitting there is much that is theoretical, this is necessarily so from the very nature of the condition. I have no doubt as to the accuracy of his observations but I think there is room for doubt in their interpretation.

W e have already had occasion to mention these incidentally, and the question arises what proof have we that Eclampsia is due to a poison circulating in the blood ?

T h e strongest proof is found in the microscopical appearances of the various organs, and their resemblance to some other cases of poisoning e.g. corrosive sublimate and carbolic as pointed out by Meyer Wirz<sup>39</sup> ; also phosphorus and lead poisoning.

Assuming that Eclampsia is due to a specific poison. Is it an auto-intoxication, or is it introduced from without?

<sup>25</sup>  
Herman favours the latter view. He discusses the similarity of Clinical symptoms between acute yellow atrophy and Eclampsia, and compares the condition of the liver in acute yellow Atrophy with that of lead poisoning.

H e further quotes Albutt's view of the marked similarity between renal disease of Pregnancy and lead poisoning and says "I can hardly doubt that the two diseases are closely allied. In both the extreme suddenness of the onset, often without the slightest warning is what we should expect if a poison from without attacked the patient "

H e then goes on to say that if any of the so called defence organs had broken down, and the patient has been gradually poisoned, we should have the symptoms appear more gradually.

A s further proof of the external origin of the poison, we have the observation that Eclampsia often occurs in what looks remarkably like an epidemic.

T h u s in Edinburgh maternity between November 1st and December 6th 36 days there were 37 confinements and six eclamptics. Kedernath Das states that in Calcutta he has often seen two or three cases together, and once five in thirty hours.

I t is a remarkable fact - if not a coincidence that the only two cases we had in Clatterbridge Hospital for the past year occurred in the same fortnight.

W e r e Herman's opinion - and he only gives it as an opinion - correct, it would also explain post-partum fits. Here neither foetus nor <sup>placenta</sup> ~~pleuria~~ can participate directly in the production of the fits, and a poison from without would explain their occurrence .

<sup>56</sup>  
According to Schauta in 185 cases fits came on during labour, in 42 they preceded it; in 82 they followed it.

<sup>14</sup>  
Dél<sup>14</sup>ore and Rode<sup>14</sup> first suggested bacterial <sup>t 31</sup> infection as the cause of the Toxaemia. Leinow<sup>14</sup>isch



examined the blood in 44 cases, and obtained 25 complete cultures of a <sup>1</sup>deplococcus, which when injected into guinea pigs produced acute anuria haemorrhagic <sup>2</sup>indometritis and <sup>3</sup>tetany <sup>ie</sup> spasms,. In other cases the germs were found in the foetal blood.

<sup>42</sup>  
Müller argued that the disease is due to bacterial infection of the uterus; as it may come on several days after labour it cannot be due to the foetus, and as it only occurs in pregnant parturient or puerperal women the seat of bacterial invasion must be the uterus.

<sup>61</sup>  
Stroganoff's arguments would also lend some support to this theory; although he claims that it is of the nature of an infection,. He avers that the disease is characterised by fever, immunity from another attack, a partiality for primiparae and marked variations in intensity. Two or three cases will often occur in hospital together, or be admitted from the same neighbourhood. To this add the statement made by <sup>12</sup>Sir Halliday Croom "What the explanation of the interesting fact of the prevalence of Eclampsia in the autumn and spring months is I cannot venture to say but that that is a fact is beyond dispute." This might also be taken as evidence of the infectious character of the disease, but unfortunately we have had the same thing said of Cancer and still we have

no further evidence of its being infectious.

### An Auto-Intoxication.

T h i s is the theory which receives most support, and on a minute examination of the facts, as related in the literature, I am more and more struck with its great resemblance to uraemia.

T h a t the fits themselves are practically indistinguishable from those of uraemia no one will gainsay and we base our diagnosis of Eclampsia upon certain clinical facts already mentioned.

*i 21*  
Freëchs was the first to bring forward the theory that Eclampsia was uraemia occurring in pregnancy and was supported in theory by Traube and Rosentstein.

T h e y however considered that in uraemia the fits were due to the poison acting in the central nervous system, while in eclampsia they were more of the nature of reflex <sup>x</sup> spasm associated with anaemia of the brain. Were their view correct it would still help us but little in the elucidation of the mystery as the cause of uraemia convulsions is enveloped in as much obscurity as Eclampsia itself.

Hektoen in his pathology adopts the theory that uraemic convulsions are due to an auto-intoxication ,

and it is on this supposition that the cause has been largely investigated in both diseases.

I n fact work has been done on almost parallel lines.

O u r reasons for considering Eclampsia to be an auto-intoxication have already been explained, and it would probably make no great difference in the appearances whether the poison were introduced from without or came from within.

Appearance of the maternal and foetal organs suggesting some toxin.

I t is interesting to note that in a case of nephritis recorded by Sikes<sup>5.8</sup> exactly the same liver conditions were found on the foetus, as in Eclampsia.

Bouchard<sup>8</sup> was probably the first to bring forward the theory of auto-intoxication. His theory was that in health while metabolism was going on, the excretory<sup>et</sup> organs, lungs, kidneys, skin, alimentary system, excreted waste and poisonous products. So long as these excretory<sup>et</sup> organs performed their duties properly, then all was well, but if for any reason there was an excessive metabolism, <sup>or</sup> ~~as~~ a deficient excretion then the result was a general poisoning of the system.

T o these organs already mentioned we should add the thyroids <sup>the</sup> parathyroids liver and supra-renals &c. for if any of these fail in their functions we have



an auto-intoxication produced e.g. diabetes, produced by pancreatic inadequacy.

Bouchard concluded that normally the urine was toxic, and proceeded to measure the toxicity of standard urine as follows :- He took the standard weight of healthy individuals to be 65 kilogrammes and the average amount of urine passed in 24 hours by such individuals as 1200 C.C. *(Quoted by L. Den. Journal of Abk)*

He estimated the urotoxic dose of such urine by injection into the auricular vein of a rabbit, and found 40 C.C. sufficient to produce death of the rabbit. Thus his urotoxic coefficient is

$$\frac{1200}{40 \times 65} = .46.$$

Subsequent observers practically accepted Bouchard's urotoxic co-efficient even though their experiments were often carried out under totally different conditions - hence the conflicting results obtained.

Bouchard himself found that 120 C.C. of distilled water injected into the auricular vein of a rabbit caused death, so that too much importance cannot be attached to his urotoxic dose of urine which sometimes exceeded this.

Bouchard's only precaution previous to injection of urine was filtration and the addition of bicarbonate of soda to neutralise the acidity.

Tarnier took the matter up with his pupils and proved that the urine of pregnant women was less toxic than of non-pregnant women. From this they concluded that the toxins had accumulated in the pregnant in greater amount than in the non-pregnant women.

*Eden* <sup>72.</sup> \* <sup>72</sup> Rosenberg found that the substances taken out of eclamptic urine by alcohol and ether were not toxic, whereas an aqueous solution was, and concluded that the toxic substance was only soluble in alcohol and ether.

Schumacher <sup>57</sup> by careful controlling experiments practically refuted all that had been done by Bouchard Tarnier and others.

He found briefly that saline solution if sufficiently concentrated was lethal; that the toxicity of the urine depended largely on its concentration, thus urine with S. G. 1020 was toxic, but if diluted to 1010 the toxicity was diminished. He also found that there was no difference in the toxicity of the urine in normal pregnancy, puerperal albumenuria or Eclampsia, . <sup>60</sup> Stewart had already satisfied himself that previous observers had obtained erroneous results owing to using septic urine, and found that on boiling the urine and injecting carefully there was practically no toxicity .

We may therefore conclude that although Bouchard's original theory is more than likely the

correct one, his methods of observation were faulty.

A p a r t from these experiments, every known constituent of the urine has been at different times injected with a view of proving or disproving their power of causing eclampsia.

T h u s it was thought that owing to the small amount of urea excreted by Eclamptics and the comparatively enormous amount after <sup>i</sup>düeresis had been established ~~that~~ there was a retention of ur<sup>e</sup>ia in the blood. Zweifel <sup>69</sup> has made many observations on this point. He shows that whereas normally 83% of the total <sup>N</sup> in the urine is in the form of ur<sup>e</sup>ia, in Eclampsia this is not so, but the worse the case of eclampsia the less the relative amount of urea to the total Nitrogen.

F r o m this we might conclude that the ammonia is gradually <sup>fixed</sup> ~~fixed~~ by an acid in a form in which it cannot be transformed into urea e.g. if the acid be Hcl. as N.H. <sup>4</sup> Cl.

<sup>han</sup> ~~Larg~~meister <sup>68</sup> considers there is an increase in the ammonia of the urine and Neumcister says there is an excess in the acid in the blood accounting for this. Thus if an animal be given an excess of mineral acid the ammonia in the urine is increased, until it actually displaces the urea.

I n this relation we may quote the experiment of ( Ecks ) fistula, between the portal vein and



inferior vena<sup>3</sup> cava, the ammonium salts are no longer transformed into urea and some of the fore-runners of urea e.g. ammonium carb<sup>am</sup>onate produces symptoms very similar to uraemic ones.

Hahn could however find no excess of this substance in the blood. (*Quoted from Bikes*<sup>58</sup>)

Moreover we have the fact pointed out by *Wicifel* that the urine in Eclampsia is not necessarily acid but may be alkaline.

From a review of the above experiments we are forced to the conclusion that so far as toxicity of urine is concerned it is still not proven; that acid poisoning may fix the ammonia and cause symptoms of Eclampsia although in many cases the urine is not acid, but alkaline.

Blood

In regard to the blood in Eclampsia, experiments were undertaken with a view to prove its toxicity

The first of these proved inaccurate as the serum produced conglutination in the right heart, and pulmonary artery, and consequently asphyxia.

<sup>16</sup>  
Doleris and Butte succeeded in extracting a crystalline <sup>in</sup>organic body from the blood serum which when injected under skin of rabbits &c caused death with convulsions.

<sup>20</sup>  
In 1894, Mairat and Bo separated an albuminoid body from the blood serum of a healthy dog which

had a similar effect. (Quoted by Eden)

Butte ( Quoted by Zweifel ) thought he could find more urea, in the blood of eclamptics, but Zweifel could not confirm his observations.

Spiegelberg considered there was an excess of ammonia in the blood, and said that he could produce Eclamptic symptoms by the injection of ammonia salts into animals.

Zweifel however denies this and says that in eclampsia there is no excess of ammonia in the blood.

With these contradictory statements it is difficult to form a conclusion, although from Massens experiment with Eckes' fistula, and the formation of ammonium carbonate, it is only reasonable to suppose that if the liver function is disordered this substance may be formed in the blood.

This is undoubtedly the line of research, which offers most hope in the elucidation of the problem of Eclampsia.

#### I n c r e a s e o f F i b r i n .

<sup>29</sup>  
Kolman and Dienst found that there was an increase of fibrin both in the maternal and foetal blood. The cause has been variously attributed to foetal metabolism, and also to the breaking down of <sup>eu</sup>leucocytes.

<sup>47</sup>  
Politi conducted some experiments on rabbits with placental extracts in order to show that in

Eclamptic placenta<sup>e</sup> there is a conculating substance, which passes into the maternal circulation causing fits.

F o r his first experiment he used the placental extract from a woman who had had severe eclamptic fits. The injection caused severe dyspnoea and death in 15 minutes.

Another similar experiment was carried out with the placental extract of a woman who had severe albumin-urea. The second rabbit had also severe dyspnoea but recovered. Still another experiment was done with placental extract from a woman who had much sickness and headache, but no albuminurea .

I n this case the rabbit had also severe dyspnoea but recovered more quickly.

T h e controlling experiment was with that of a healthy woman, and was found to be less marked, but still severe dyspnoea <sup>but</sup> as death was due to clotting in the right heart, we may conclude that the experiments are practically valueless.

A s we have already seen, normal saline solution may cause death in the same way, when 120 C.C. are injected however slowly by causing severe dyspnoea and spasms; moreover one rabbit may stand more than another rabbit of equal weight a good deal depending on the vitality.



I t is especially to be noted that severe dyspnoea was the prominent symptom in all the rabbits injected so it was really a difference in degree.

Numerous other observers have experimented on the same lines, amongst whom may be mentioned, Ascoli, Poter<sup>ei</sup>, ~~W~~ Richardt, but the results of their experiments are not very convincing if we remember that the blood serum of one animal is often lethal to another by causing conglutination, and even apart from this OEDEMA of the BRAIN has also been assigned as a cause of Eclampsia and acting on this theory Dr. Arthur Helme<sup>24</sup> has treated cases by puncture, and drawing off cerebro-spinal fluid. That his case was successful is - I am afraid - no evidence of the truth of the theory, as witness Dr. Ballantynes<sup>3</sup> case performed most carefully but the patient died,. Moreover as Herman remarks we often have cases of Eclampsia where there is no general oedema, which one would inevitably expect had this been the cause .

<sup>also.</sup>  
T h i s is contradicted by Hektoen in his reference to uraemic convulsions.

#### N e r v o u s I r r i t a b i l i t y .

M a n y writers suggest that Eclampsia is due to what one might term "Nervous Irritability" The brain as it were is in a state of nervous irritability and the advent of labour with uterine contractions tends

to augment this state with the result - Eclampsia.

I n the fifteen cases which I have attended  
most of the patients were well known to me, and as far  
as one could judge there was no excessive nervous  
disposition .

I n fact the disposition of some might be  
described as placid.

Moreover in one case which I know very well,  
whom my partner Dr. Yeoman attended in two confinements  
she was the subject of severe Epilepsy,. Had brain  
instability any effect on the determination of Eclampsia  
one would have expected fits during her pregnancy.  
Such has not been the case, and in fact since nursing  
her last child - now eighteen months old - she has  
never had a fit. This bears out the experience of  
elderly practitioners who maintained that pregnancy  
cured Epilepsy . It might have been a risky experiment,  
but this particular individual married on the  
advice of an old practitioner who maintained it often  
cured the condition. It may simply be a case of  
"Post hoc ergo propter hoc" but she is very satisfied  
to think otherwise.

Xan  
~~Ker~~thin, Peptones, Globulins ( so called *Leucomaines*)  
*acetone* have all been investigated with a view to  
proving their responsibility in the causation of  
Eclampsia but with no certain result. Some of these

have been found on injection into rabbits to cause convulsions - see Schäffer on *acetone*. Unfortunately we have so many substances which do this that it is difficult to arrive at a conclusion as to the offending one.

O n reviewing the evidence as to the cause of Eclampsia, one is especially struck with (A) the evidence adduced of its being a poison introduced from without.

(B) W i t h its being an auto-intoxication and its similarity to uraemic convulsions .

I n support of the first contention we have (1) the season of the year at which the greater number of Eclamptic cases occur, (2) the fact as already pointed out of several cases occurring together (3) the fact that the greater incidence is on primipara, (4) the occurrence in the puerperal state, when neither foetus nor placenta can very well participate, (5) The immunity conferred as claimed by Stroganoff (6) The sudden occurrence of the fits - as pointed out by Herman - often without any premonitory or pre-eclamptic symptoms. (7) The similarity of the condition to acute lead poisoning.

T h a t it is an auto-intoxication We have the similarity of the fits to those of uraemic convulsions.

T h e latter we know to be an auto-intoxication



due to kidney disease, although we are quite ignorant of the exact determining cause.

The similarity of the fits in both cases is very marked while in both we have a diminution in the amount of urea passed. The albumen would only seem to be an indication of the presence of the disease in both cases, as it is well known to be often absent in cases of chronic kidney disease.

<sup>69.</sup>  
According to Zweifel we have a marked reduction in the oxidised sulphur in both conditions showing that there is deficient oxidation in the tissues.

Then in both Eclampsia and in some cases of albuminurea we have characteristic changes in the organs of the child ( Sikes ).

It is true that these only indicate the presence of a circulating toxin or poison in both, but still the similarity is significant.

In uraemic convulsions we have the presence of urea in the sweat glands, bile and alimentary canal, and I have no doubt this is an effort of nature to get rid of the offending poison.

T h e evidence of urea being the cause has already been discussed.

W e have finally the similarity of treatment adopted in both conditions ~~in~~ ~~to~~ ~~Bright's disease~~. Rest and milk diet with the object of lessening the amount of urea and the work of the "Defence Organs" / and the same treatment <sup>is also adopted</sup> in cases with well marked pre-eclamptic symptoms.

I do not propose to decide - as I consider it an impossibility with our present knowledge whether Eclampsia is an auto-intoxication or an Intoxication due to some poison introduced.

S u f f i c e it to say that it is undoubtedly an Intoxication, and this is borne out by the results of treatment.

T h a t the treatment is variable cannot be denied, but that good results have accrued from methods of treatment specially directed either to dilute the poison in the system or to get rid of the poison through the kidneys skin alimentary system is equally certain.

## INJECTION of SALINE

<sup>27</sup>  
Dr. Jardine has adopted this method extensively in Glasgow and has reduced the death rate from 47 % previous to its introduction to 17 % for the last three years. He explains its use by saying that the benefit is due to the diuretic effect produced, and to the elimination of urea and uric acid. At first he used bicarbonate of Potash in order to stimulate diuresis, but later he resorted to ac<sup>e</sup>tate of soda 1 drachm, sodium chloride 1 drachm.

T h i s change was suggested by Professor Stockman, as the Potash is well known to have a depressant action, and cause convulsions. He incidentally remarks that the Potash might neutralise the poison. This is quite possible if we accept the theory that Eclampsia is due to acid poisoning. The mode of action of these bodies is probably by changing the composition of the blood. Starling points out in the article of "Urinary Secretion" in Schaffers Physiology that the easiest way to excite the flow of urine is to alter the composition of the blood through the administration of large quantities of water or of certain drugs known as diuretics. Ac<sup>e</sup>tate of Soda and Common Salt belong to this last group and Starling says if these bodies be injected into the blood, a very copious



secretion of urine is soon evoked, even if previous to the injection the secretion had been at a stand still.

I t has been found that their diuretic properties are proportionate to their power of attracting water, and as soon as they reach the blood they have the power of drawing water from the tissues to the blood hence dilating the blood vessels and expanding the kidney itself. This physiological action explains their benefit in eclampsia, where the urinary secretion is brought to a stand still.

I n the withdrawal of fluid from the tissues it is possible that poison is also withdrawn from the tissue passed on to the kidney and excreted with the urine.

B e this as it may it is certain that the urea and uric acid are markedly increased and that there is often great benefit to the patient and <sup>in \*</sup> ~~Lunbeck~~ has shown that their diuretic action is proportional to their osmotic pressures. As already stated they produce a hydraemic plethora and the secretion of urine is co-terminous with this hydraemic plethora.

I have used saline injection in two cases of Eclampsia with marked benefit. In the first case, Mrs. H it was combined with accouchement

force. History of the case is briefly as follows :-

I n November 1901 I was called to see a Multipara aged 30 suffering from severe fits, all her previous confinements had been normal. I arrived and found she had complained of headache and sudden blindness and almost at once had a fit. These fits had followed one another in quick succession and although she was at full time, on examination, the os barely admitted one finger. I transfused two pints of normal saline as the urine was very scanty. In two hours after the transfusion she had passed a considerable quantity of urine, but in bed.. After the passage of the urine, the fits still went on, and my custom then was to deliver every case of Eclampsia as soon as possible, if at full time. My partner gave chloroform while I dilated the os with my fingers and delivered an apparently still born child, the operation taking twenty minutes during which time she had three fits. The child gasped once, but every effort on our part failed to resuscitate it any further.

T h e Mother made an excellent recovery and had no fits after the birth of the child.

T h e urine was free from albumen on the tenth day after delivery. Specimen taken by Catheber.

Urine passed and previous to labour loaded with albumen and very scanty. That the saline caused

the flow of urine, I felt quite convinced, as I got the nurse to draw off all that the bladder contained on my arrival, and the fact that she passed it in bed prevented any <sup>est.</sup>intimation of quantity of urine or a -  
mount of albumen.

S h e had no other drug except the chloroform administered by my partner Dr. Yeoman. Since her confinement, now nearly five years ago, she has had no more children, simply because I could not guarantee that she would not have a return of the fits.

T h e other case I treated was a patient admitted to Clatterbridge Workhouse - really a tramp- December 1902. She was a primipara and when seen by me was already in labour. The fits were never very severe, but on arriving, I enquired about the bowels got the nurse to inject a pint of normal saline solution. I then gave her chloroform, and as the os was fairly well dilated applied forceps and delivered her.

S h e had three fits after delivery but made a very good recovery. She passed within four hours after delivery 40 ozs of urine.

N o w this might have happened in any case, as it is a well known fact, that after cessation of the fits very free diuresis takes place. However I think that the absorption of the saline solution from the rectum and the passage into the circulation was



a probable factor in her quick recovery and very free diuresis. My reason for injecting saline into the bowel in this case was due to the fact that I had no apparatus with me for giving it otherwise. The objection to this method brought forward by some Obstetricians that it raises the blood pressure and hence makes Eclampsia worse is not a valid one.

Herman states that we have no proof for stating that high blood pressure is a bad thing in Eclampsia, and quite apart from this if we are to have free diuresis we must either raise the blood pressure or alter the constitution of the blood, and this method does both.

I n July number (Brit: Obstet: Journal 1905)

Jardine gives an account of fifteen cases admitted to Glasgow Maternity, and treated by saline solution and acetate of soda. These cases all recovered and it is worthy of note that he used in addition the hot pack, free purgation and *venesection*. He says "one need have no fear to bleed freely if the saline is immediately injected". Another point of note is the fact that he injects the saline directly into the vein, instead of into the loose cellular tissue of the breast as formerly. His results have been so uniformly good, as to compare favourably, if not to surpass any other method of treatment.

## THYROID TREATMENT

A p a r t from theory of Thyroid Inadequacy  
<sup>43</sup>  
Nicholson states his reasons for giving thyroid to  
Eclamptics.

"A substance which so powerfully stimulates  
"both metabolism and elimination, increases largely  
"the secretion of urine and the excretion of urea  
"and other urinary bodies, acts as a specific vaso-  
"dilator<sup>s</sup> thus leading to perspiration and lowering  
"of the blood pressure cannot fail to prove a potent  
"means of safeguarding the pregnant woman from the  
"effects of toxic<sup>x</sup> poisoning".

T h a t this statement of the case is the  
correct one probably no one will deny and it was on  
the strength of this that I have given it, as oppor-  
tunity arose. My experience with thyroid in the  
treatment of Eclampsia amounts in all to three cases,  
and while they all three recovered I shall content  
myself with the narration of one case, which I treated  
in Clatterbridge Hospital, and accordingly had better  
opportunities of noting the effect of treatment.

Mrs Norman 37 years multiparae sent in by Dr  
<sup>n</sup>  
Knott of Sutton on April 3rd 1904 suffering from  
Eclampsia.

H e gave me the history that her previous

confinements had been normal, and she was now nine months pregnant.

S h e had developed Eclamptic fits on the morning of April 3rd. and on testing her urine, he found it almost solid. The only treatment he carried out was the injection of pilocarpin.

I saw her at 10 p.m. on the 3rd. after admission, and she was all but unconscious; she could be roused with great difficulty although she had had no fits since her admission 2 hours previously.

Her face hands and feet were *oedematous* and her pulse was slow and full 60 per minute.

I asked the nurse to give a soap and water enema, using plenty of water, and after the bowels were thoroughly cleared out, I roused her sufficiently to make her swallow 15 grains of thyroid, which were broken up in milk.

A s Dr. ~~Knott~~ informed me she had passed only about 10 ounces of water during the day, I told nurse to measure carefully all she passed.

S h e remained fairly quiet all night and passed 12 ounces of urine during the night, but had no fits - Nurses description was "unconscious but woke up occasionally to consciousness when I fed her with "milk".

APRIL 4th. Woke up and had three fits in the morning



and she had a typical one as I saw her just at 9 a.m.

I then determined to try and get the kidneys to act, as she had passed so little urine, and thought of giving her thyroid rectally. I mixed up 5 tabloids (25 grains) in all with a small quantity of milk and awaited a favourable opportunity as nurse informed me she could swallow. She swallowed the milk at 10 o'clock, and then seemed to go off in an unconscious stupor.

A t 12 o'clock she had another fit which lasted - according to nurse -  $1\frac{1}{2}$  minutes.

A l l this time temperature was  $100^{\circ}$ . Nurse was able to wake her up for nourishment, and she had no more fits on the 4th. Amount of urine passed on the 4th. 14 ounces and almost solid with albumen.

S h e had a fairly quiet night and no fits, but only passed 4 ounces urine.

APRIL 5th. Temperature had now fallen to  $98.2^{\circ}$ . and pulse was 70.

S h e took a fair amount of milk during the day, had 15 grains of thyroid and had only one fit. Notwithstanding she remained in an unconscious state and could be aroused with difficulty.

Quantity of urine passed for April 5th and the night of April 5th 35 ounces.

APRIL 6th. Had three fits in the morning, and was given

30 grains of thyroid in milk.

Shortly after this pulse rose to 89 and temperature 98.8. Bowel was washed out in the afternoon. In the evening had two more fits, pulse was now 90. During the day she passed 40 ounces of urine, but still there was no perceptible diminution of the oedema. She had had 4 pints of milk in the last 24 hours.

S h e passed a fairly quiet night with no fits urine passed 5 ounces.

APRIL 7th. Had two more fits in the morning, and took 25 grains of thyroid, she had another fit in the forenoon so I gave her other 10 grains.

H e r coma seemed to have deepened, but the only great change was noticed in the pulse now 110 and the flushed face. She passed 90 ounces of urine during the day. During the night she passed 30 ounces of urine and in the morning the oedema was distinctly less.

APRIL 8th. The membranes ruptured at 6 a.m. and I told the nurse not to give any more thyroid as I considered she was suffering from "Thyroidism" the pulse was quick face flushed and she was very restless.

T h e os was dilated about size of two shilling piece and on attempting to dilate still further she had a very severe fit, so I determined to leave her

alone and await events.

During the day she passed 75 ounces of urine - night 15 ounces - albumen copious. She had the bowel washed out in the afternoon.

APRIL 9th. Child born dead at 5 a.m. and I was sent for as nurse could not expel placenta.

I arrived at 6 a.m. expelled placenta with little difficulty and found her brighter looking than I had seen her. She could answer questions but almost immediately relapsed into a semi-conscious state.

During the night she had passed 10 ounces of urine.

H e r pulse was now very rapid about 120 per minute and her temperature 100 ~~Fahrenita~~ Fahrenheit. I thought she had too much thyroid. I left at 8 a.m. and was surprised to be sent for at 10 a.m. in a great hurry. She became quite unmanageable - so much so that two nurses could not hold her in bed, so they had to put her in a canvas jacket generally employed for dangerous patients.

W h e n I arrived I got the jacket off, but had to put it on, as we could not keep her in bed.

T h e contrast was most marked - the patient who the day before lay almost lifeless now suffering from acute mania.

T h i s continued for 24 hours, and although



she passed urine it was quite impossible to collect it.

April 10th.

Towards evening she became much quieter and the jacket was removed. The temperature was now 100.6 and the pulse 110. From now onwards she made practically an uninterrupted recovery, and she left the Hospital on May 1st.

F r o m April 10th the urine was markedly increased for the first week, but afterwards became normal in amount, and when she left there was just a faint trace of albumen.

I have communicated with Dr. Knott Sutton since and he tells me she is perfectly well.

T h i s case is of interest in many ways.

Herman states that most cases of Eclampsia - and it is the general experience, run their course in 48 hours.

T h i s case went on from April 3rd to April 9th the last fit occurring on April 8th i.e. 5 days.

D u r i n g this time she had in all 120 grs. of thyroid.

I t is worthy of note that the great increase of urine occurred only with the advent of the symptoms of "Thyroidism" and on April 7th when she was undoubtedly suffering from Thyroid poisoning

she passed 120 oz. of urine in 24 hours. It may be dangerous to push thyroid to such an extent as this, but in this case at least it seems to have been quite justified.

T h e symptoms of acute mania passed off in 24 hours, and though rather alarming at first left no evil after effects.

I t has occurred to me over and over again since. Would this woman have died if she had not had thyroid? It is quite possible that the prolongation of the case was due to the thyroid given in large doses, which kept the kidneys fairly active.

H a d thyroidism been produced more quickly, we might have expected a quicker recovery.

F r o m the appearance of the foetus I should think it had been dead at least 4 or 5 days, but of this I am not certain.

A n o t h e r objection might be raised that this was not a case of Eclampsia "proper" at all, but of chronic kidney disease. Against this is the fact that the albumen cleared up after delivery, and that she is now - two years after - perfectly well.

T h i s illustrates what I said earlier in the Thesis that before Thyroid can be of any

curative value in Eclampsia we must have the pathological effect of the drug.

<sup>62</sup>  
It is interesting to note in this connection that Sturmer has treated 43 cases in the last two years by this method with a mortality of 12%. Previous to this form of treatment his mortality in India had been as high as 46% although it on one occasion fell to 13%.

He also uses morphia and saline infusions as adjuncts, but pins his faith to thyroid.

He remarks that after giving (30 - 40 grs) in 24 hours there is a marked increase in the flow of urine, and this bears out my experience that large doses are necessary.

<sup>38A</sup>  
Mac.Nab also reports a case of Thyroid given (75 grs in one dose) with recovery, but as he had also given morphia chloral and Bromide and saline, it is difficult to form a conclusion in regard to any one drug in this particular case.

Ballantyne has also used it, and as Berkley<sup>6</sup> puts it "Thinks well of it".

In fact it is indirectly to Ballantyne that we owe the thyroid treatment of Eclampsia, as it was this careful observer who first brought Langes cases to Nicholson's notice.



## P U R G A T I O N .

25  
Herman does not believe in Purgation and quotes Sutton as saying that he believes purgation does harm in uraemia. He also says "If I knew of a condition in which purgation cures convulsions then I would advise their use. Now if we admit that the elimination of urea is beneficial, purgation is one of our most valuable assets in the treatment of Eclampsia.

I t has been abundantly proven in uraemic convulsions that we have an excess of urea in the bile and alimentary canal. This is supposed to account for the diarrhoea prevalent in Brights disease, and probably would also exist in Eclampsia, were it not due to the torpor produced by the fits. An old practitioner once told me that he never despaired of Eclamptics if he could get the bowels moved, and those cases which most frequently died had no action of the bowels.

Moreover any one familiar with diseases of children knows that convulsions can be set up by intestinal irritation. If you clear out the irritant with a brisk dose of castor oil, you can often cure convulsions in children. The probability is that the same thing holds good in Eclampsia, and just as urea is

excreted by the intestine, so also some more patent agent may be excreted and got rid of if we can only get the bowels to move.

### Vena<sup>e</sup>l section.

This is advocated by many as reducing the amount of the poison, and granted that the poison is circulating in the blood, it must reduce it in proportion to the amount of blood extracted. On this supposition, Blacker Lockyer Fothergill Munro Kerr and Russel<sup>6</sup> (Quoted by Berkley) bleed and then follow by injecting with saline infusion. This treatment has certainly a great deal to recommend it, and is both scientific and calculated to do good. The only objection that could be offered, is the blood of more importance to the patient than the saline? If there is a haemolysis as maintained by many observers, it is possible that we deprive some patients of their very life blood by bleeding.

My only experience of bleeding was in one case, practically in Extremis - when I opened the Median basilic vein, but could not get her to bleed. The fits started 1 $\frac{1}{2}$  hours after delivery at 2.15 p.m. and she died at 12 p.m. In this case Crobon Oil failed to move the bowels and vena<sup>e</sup>l section had no effect. Simpsons Steam bath was also given without effect. Had I such a case now-a-days I

should use saline injection and thyroid combined.

The most striking benefit from bleeding known to me was told me by R.D.R. Allison M.D. of Liverpool to whom I am indebted for the notes.

"When in Oldham assisting the late Dr. <sup>Corn</sup>~~Leon~~ in 1891

I was called at 2 a.m. to a woman having fits during labour. I found her a primipara aged 20 in labour at full time and with well marked Eclampsia.

The ~~os~~ was not sufficiently dilated to use forceps so I administered Chloroform, and kept her more or less under its influence till 6 a.m. when I delivered her of a living child by means of forceps I administered chloral hydrate by the rectum, and <sup>..</sup> m II Crobon Oil by the mouth. The Crobon Oil did not move the bowels, and I observed no benefit from the Chloral Hydrate, I passed a <sup>t</sup>Catheter and drew off some of the urine which I heated in an iron spoon over a gas jet, and the urine became almost solid.

From beginning of labour, there had been 6 severe fits, and the patient was now becoming so cyanosed that I withdrew the anaesthetic until the colour improved, the fear of asphyxia deprived me of pushing the anaesthetic just when she required it most. After the delivery at 6 a.m. the convulsions became less frequent until 8 a.m. but they then came on more frequently than before, unconsciousness



became complete, and the case looked hopeless. At 9 a.m. I went home for breakfast thinking the patient was dying when in my absence she suddenly stood up in bed, and severe hæmorrhage set in. This rendered her almost bloodless, and when I came she was blanched from loss of blood, but the convulsions had ceased. I at once injected 1 pint of Saline solution bandaged her limbs and gave ether and brandy hypodermically. Patient became conscious at 4 p.m. and made a good recovery". This is the experience of Dr. Allison - a brilliant student at Edinburgh University and an able practitioner. He carried out as it were by accident the treatment which many practitioners now adopt as a routine.

#### H o t   W a t e r   B a t h s .

T h e s e can only be used if the patient is not in labour, and if not too hot would seem to act by allowing a greater amount of blood to come to the surface of the skin, and so enabling the skin to do the work of the kidneys. Herman thinks it is useful in cases of Coma, if the pulse is beginning to fail to put the Patient in a bath of 102° Fahrenheit, and keep her in for half an hour. This would probably help by stimulation especially if the temperature were subnormal.

T h e Hot pack has perhaps the advantage that

it promotes free perspiration. Its action is practically the same as that of a poultice. The patient is wrapped in a sheet wrung out of very hot water and then surrounded by a macintosh. That this makes the skin act almost when nothing else will, anyone who has had experience of the Moist room in a Turkish bath can test for himself.

G o in to a room with hot air provided it is a dry heat and most people can stand a temperature of  $200^{\circ}$  but if the air is moist  $120^{\circ}$  makes one perspire so freely that it is impossible to stand it long. Principle of Conduction - The Hot water pack owes its great efficacy to the fact that the patient perspires freely, and gets rid of the poison in this way.

#### C o l d B a t h .

T h i s is really employed to reduce temperature in cases of ~~Hyperpyrexia~~ <sup>Hyperpyrexia</sup>. Herman strongly advocates its use where the temperature rises to  $106^{\circ}$  or  $108^{\circ}$ .

T h e patient should be placed in a tepid bath  $70^{\circ}$  or  $80^{\circ}$  Fahrenheit left in for 15 minutes, taken out rolled in blankets and allowed to perspire. This method has been largely used in the ~~Hyperpyrexias~~ <sup>Hyperpyrexias</sup> of different fevers, and their advocates claim great benefit from them.

## DRUG TREATMENT .

I n addition to the methods already described we have the drug treatment, which is generally combined with one or more of the other methods.

### C h l o r o f o r m .

I n regard to the treatment of Eclampsia we owe a great deal to Berkley who sent a circular to our chief authorities, in order to ascertain the different treatments adopted. The result is published in the Journal of Obstet. & Gyn *of Brit. Empire* March 1904. I shall first discuss Chloroform. Chloroform.

M o s t if not all Obstetricians use Chloroform in Eclampsia, if not to quieten the patient during the fit in order to put her to sleep during operative measures.

T h e giving of Chloroform to control the fits themselves is of doubtful value, indeed we may only be putting off the evil day by giving chloroform, for although it controls the fit, it does not in any way favour the Elimination of the poison. This is at least the view taken by Herman and he thinks it is really no use in controlling the fits unless the patient is kept under all the time. If the latter course is adopted, we have the danger of



producing Chloroform poisoning. This is well illustrated in Allison's case where he says "I had to withdraw the anaesthetic when the patient most required it, for fear of producing asphyxia". Those who use it for controlling the fits maintain that it does so, and at the same time lowers arterial tension and lessens venous congestion.

S i r J. Williams Champneys & Tate use it if fits are frequent.

Wright uses it exclusively.

Blacker Fothergill Lockyer and Russel use it till Morphia has had time to act.

#### M O R P H I A .

Morphia is a drug which is largely used in the treatment of Eclampsia, and in the hands of some with excellent results. <sup>66</sup> Veit gives as much as 3 grs. of morphia in seven hours, until the patient is deeply asleep. He records 60 cases treated in this fashion with only two deaths and attributes its failure in the hands of others to the fact that they do not push it sufficiently. This was tersely answered by Dr. Corby at the British Med: Assn: Leicester 1905, when he remarked "I myself pushed the morphia treatment, and am afraid I helped to push one or two of my patients into another world:"

<sup>73</sup>  
De la Harpe has published the statistics of

the Rotunda Hospital Dublin since the introduction of Veit's Morphia treatment. This takes us back 13 years, and for this period the mortality has been 16'9% as compared with a mortality of 35'3% before its introduction. Although reduced by 50% this mortality does not compare favourably with Veits 60 cases and two deaths or Jardines 15 cases and no death. We however have to make allowance for difference in locality, as has already been referred to in this Thesis.

Personally, I have used morphia in only one case in the Simpson Memorial Edinburgh in 1897, and administered 1 gr: ~~only~~ She made a good recovery.

I n the light of thyroid and Saline treatment it is not a little difficult to understand the action of morphia. At one time the teaching was against giving morphia in kidney disorders, but Dr. Stephen Mackenzie and others give morphia in uraemia and also in heart conditions, where it was previously unheard of.

T h e striking fact about morphia and thyroid is that their best results are obtained by producing practically poisonous effects, and as the two drugs are pharmacologically almost antagonistic in action, theoretically, it is difficult to understand their beneficial effect. Nicholson suggests that they have vaso-dilator action is common, and if the benefit accruing from this, more than counterbalances the inhibitory effect

of Morphia on Metabolism, we may have the explanation  
C h l o r a l   H y d r a t e .

T h e most striking figures in relation to  
this drug are given by Charpentier<sup>10</sup>.. He collected  
239 cases and found when chloral given alone death rate  
was only 4'3% and when in combination with other meth-  
ods 8'5% Charpentier gives  $\frac{1}{2}$  ounce in the 24 hours -  
chiefly per rectum.

Personally I know of a case where a well known  
medical man administered two drachms of Chloral per  
rectum to a patient who had been delivered and was  
very restless. He was sent for afterwards only to  
find that she had slept so pre<sup>f</sup>oundly that she never  
awoke . This happened 10 years ago, and since  
probably through prejudice - I have never prescribed  
it.

T h e great difficulty seems to be the dosage,  
as it decomposes in the blood into Chloroform, and in  
some cases with disastrous effects.

V e r a t r u m   V i r i d e .

I t has been largely and probably beneficially  
used in Eclampsia, but as sometimes happens even with  
good remedies, seems to be falling into disuse..  
Stephenson is its warmest advocate and quoted by  
Berkley<sup>6</sup> says it "Modifies and relieves more directly  
and beneficially than any other drug, & I have known



the Vascular derangement<sup>t</sup> that endangers life" The drug lowers blood pressure by dilating the arteries and depressing the heart - hence the chief reason for its exhibition.

#### P i l o c a r p i n e .

I s another remedy which has much to recommend it from a theoretical stand-point, but unfortunately has great disadvantages - disadvantages so great that its administration is often followed by dire results. It is used from its power of producing free secretion but unfortunately the lungs participate in the general action, with the result that oedema takes place.

C o u l d we so adjust dosage as to stop ~~short of~~ <sup>dyspnoea.</sup> ~~dyspnoea~~ and evil pulmonary effects then it might be a valuable remedy, but as this is impossible, it is better to leave it severely alone.

I n dealing with the Drug treatment of Eclampsia, I do not pretend to give an exhaustive account of all the different remedies used in this connection. I have limited my observations to those which have been in general use for some time and either found or considered to be highly beneficial. There are many other drugs which have had their advocates, and I shall only mention one Iodide of Potassium, A. R. Simpson, <sup>59.</sup> whose mind was always alive to the very latest in

literature, used it with beneficial effects in a case of Eclampsia in Edinburgh Simpson Memorial. He quotes its use by a Danish Veterinary Surgeon in Eclampsia in Cows. I spoke to our Veterinary Surgeon of its use and to my surprise he said it was the only drug he used in such cases and it had a remarkable effect. Nicholson thought its effect could be traced to the thyroid and that the Iodine was picked up by the thyroid and the latter rendered consequently more active. It is also possible to explain its action from the presence of the Potash, and as this is a most active diuretic, it is more than probable that the good effect is simply the diuretic effect.

I n reviewing the work done in the treatment of Eclampsia, one cannot help being struck with the results obtained by Jardine and others. Although the remedies are not new the reduction in death rate under the new methods of employing them compares favourably with the reduction in death rate by the antitoxin treatment of Diphtheria. Thus in the Rolanda with Veits Morphia treatment we have mortality diminished 50% and in the last 15 cases recorded by Jardine not a single death.

T h e balance of evidence in the treatment of Eclampsia would point to the use of thyroid, morphia and lastly the most scientific method ad

vocated by Jardine-free purgation, the hot pack, venae-section and saline infusion. That Morphia does good by inhibiting the convulsion is scarcely conceivable; we might as well argue that it cures appendicitis by relieving pain.

U n l e s s we can clearly show that the fits themselves are the cause of death then the benefit of Morphia in this direction remains to be proved.

I t is far more rational to suppose that Morphia cures by relaxing arterial spasm and promoting free diuresis, in the same way as thyroid salines &c., We have got the poison in the system, and our main object is to get rid of it, and if we can do so, we will cure our patient. The treatment of Eclampsia is Empirical, and it is argued by no less an authority than Herman, <sup>25</sup> that while it is so, and the pathology is obscure, no real scientific <sup>or drug</sup> treatment can result. I deny this in toto.

O u r most successful drug treatment is empirical. Take for instance, Syphilis. The Organism has never been definitely discovered, and were it discovered and the exact pathology clearly defined, I doubt if it would lead to the discovery of a more beneficial drug than Mercury given either intramuscularly or by <sup>in</sup>unction. The same also applies to Malaria, and I venture to mention puerperal Toxaemia. Quinine given



in Malaria has not been rendered more efficacious from Ross and Low's brilliant discoveries. We give the remedy, the effect is noted, and it is only by careful observation in the human Laboratory that the most accurate results are obtained. Brilliant pathological research only confirms—in many instances — what has already been done.

B o t h from a scientific point of view and from the results obtained, Jardine's method is the one that recommends itself most. It is devoid of danger, and his success is due to the fact that all his efforts are directed to getting rid of the cause of the fit.

#### OPERATIONS .

In regard to operation<sup>ve</sup> interference in Eclampsia, opinion seems to have changed very considerably. In the case I have described as treated by thyroid extract, had such a case occurred *five* years previously, I should not only have felt it my duty, but considered myself almost criminally negligent if I had not delivered as soon as possible. However in the light of Herman's Statistics that the mortality is greater in cases of interference than in cases left alone, there is ample food for reflection.

#### PREMATURE LABOUR .

U n t i l within the last few years it was

generally supposed that if the mother was delivered the fits would in most cases cease. I think in this country specially, Herman<sup>25</sup> by a very careful computation of cases shows that

The fits continue after delivery in 5.2%.

The fits stop on delivery in 47.5%.

Barbour's case has already been referred to where <sup>all</sup> ~~N~~uminuria rapidly diminished and <sup>o</sup> ~~ped~~ima quickly disappeared on the death of the <sup>oe</sup> ~~f~~etus in utero, and he quotes cases from Underhill MacLaren and Spiegelberg showing the same result. Numerous other instances could no doubt be cited where the fits ceased on delivery, which amounts to the same thing, but Herman argues that they do not cease often enough to justify operative measures in fact he says "The practical importance of these figures is that they show that delivery does not influence the course of Eclampsia favourably".

He further adds that after referring to different methods of accouchement force "Such measures have no justification unless immediate delivery greatly benefits the patient, and it does not".

It is also noteworthy that Jardine in the series of fifteen consecutive successful cases already referred to says "In the obstetrical treatment of the cases it will be noticed that immediate delivery

by accouchement force" was resorted to in the earlier cases in the series and that most of the others were allowed to deliver themselves or forceps was applied after the os was fully dilated. After a very extensive experience I have come to the conclusion that delivery by accouchement force is not necessary except in cases where in spite of treatment, the fits continue to occur". He finally adds "The results in the other cases (i.e. those left alone) were equally good and the patients were not subjected to the risk of forcible dilatation of the cervix".

This further seems to indicate that the trend of opinion is in the direction of leaving the delivery of Eclamptic cases to nature.

In March 1904 in answer to Berkley's Circular, I find that Jardine replied, that if labour had not come on he would leave it to nature unless fits severe, but if labour had come on he would use manual dilatation or Bossi's dilator with forceps or version.

I am aware of the fact that the question is a most debatable one, but have gone into it more with the view of showing that at least a section of opinion has veered round to Hermans view. A certain proportion and I am not sure the greater proportion still induce labour and I may only mention Sir John Williams Eden, Fairbairn, Croft, Newnham, Haultain (if before



six weeks from term) Stephenson, Byers, Gibson, Kinkhead, Campbell, Jardine and Ballantyne (the last 3 only if fits severe)

Croom<sup>12</sup> in an article published in the Obstetrical transactions of Edinburgh, after briefly reviewing the different theories of the cause of Eclampsia, says "Any treatment which will anticipate labour avoid labour or shorten labour would seem theoretically to be the best in the long run for the patient but alas! facts do not bear this out". He goes on to say. "In view of what I have said it is somewhat remarkable to find that the pronouncement of the International Congress in Geneva was that "Wherever it is possible it is advisable to wait until labour sets in spontaneously and to complete delivery as far as possible without any artificial help &c".

O n e observation quoted by Croom struck me forcibly and that was "Dahrssen found that Eclampsia ceased more frequently after the artificial than after spontaneous <sup>v a</sup> ex~~a~~cuation of the uterus and the mortality in both cases does not differ to any marked extent". A priori one would have thought that the artificial manipulation would have tended to aggravate the condition, and of course this is possible although it ceases afterwards. This observation would tend to negative the nervous theory of the origin of Eclampsia

T h a t rapid delivery is desirable in some cases where the mother is practically in <sup>lab</sup> ~~lab~~orems, and a living child is desired, there can be no difference of opinion. The only difficulty is to decide when such a condition arises, and this must be left to the Judgment and experience of each practitioner. In Sir Halliday's Crooms two cases, such was the condition and he adopted Caesarean <sup>s</sup> section as affording most rapid delivery, and giving mother and child the best chance.

T h e s e are the most severe cases and we have got to decide between forcible dilatation of the <sup>uter</sup> ~~cervix~~, by means of manual or some form of instrumental dilatation and operative measures such as Dührssens method by circular incision and splitting cervix or Caesarean section.

W h i c h method is adopted depends a good deal upon the obstetrician, and while many would have no hesitation in using Bossis dilator or Barnes bag, they would hesitate to perform Caesarean section, which would in many cases be the wiser plan.

T h u s in Crooms cases he had intense rigidity and hypertrophy of the cervix in one case, and in the other complete closure, so that dilatation by means of metal instruments would involve severe laceration.

Personally, I should have employed Dührssens

method, but that is simply due to the fact that I am more familiar with the vaginal method of dealing with the uterus; moreover in both methods skilled assistance is a "sine qua non"

T h e only other operative measure for the relief of Eclampsia which I shall refer to is the method of

### L U <sup>M</sup> ~~N~~ E B A R P U N C T U R E

Hilme was the first to perform the operation in this country, and his case although a particularly severe one which had resisted all the well tried remedies recovered after lum~~bar~~bar puncture I have already discussed the theory of increased cerebro-spinal tension which is the rationale of treatment, and it will be sufficient at this stage to mention its supposed existence, and that the lum~~bar~~bar puncture of the ~~Arachnoid~~ <sup>Arachnoid</sup> removes some cerebro-spinal fluid and relieves the tension.

<sup>3</sup>  
Ballantyne in an Article in the (Ed: Obstet transacts) records a case of his treated in this way. In his case only a few drops of cerebro spinal fluid came away. He then dilated with Bossis dilator and delivered. The patient died 3 days afterwards remaining unconscious all the time.

Ballantyne with his usual accuracy has collected twenty one reported cases of this method of



treatment with five deaths, and as he remarks " A degree of mortality not differing much from that recorded in the Annals of Eclampsia."

From <sup>at</sup> which we have noted in regard to the treatment of Eclampsia, the prognosis would seem to be most favourable in those cases in which surgical or instrumental treatment was not called for. In fact the Surgical treatment of Eclampsia if adopted in a case where saline infusion ~~venal~~ section, thyroid extract had not been tried would seem to be a mistake. The general concensus of opinion of the Geneva Conference would seem to be the correct one, but this, time alone can decide.

T h a t great strides have been made in the treatment of Eclampsia will only become evident with the general adoption of the treatment giving the best results.

T h a t this adoption of one definite form is becoming more general is evident from the numerous obstetricians who now-a-days use saline infusion.

I n 1897 when in the Edinburgh Maternity we did not use it in a single case. However since the publication of Jardines successful cases we now find almost daily recorded cases of this method of treatment.

I t is true that generally speaking we are

inclined to publish successes, but to my mind this is only further evidence of the advantage of this method and its increasing use. ~~I~~ I mean the number of successful cases published. That thyroid extract may have a certain sphere of usefulness, I do not for a moment deny, and I have used it as already related in 3 successful cases, but the great objection to it is the fact that we have to produce "Thyroidism" in an already debilitated patient.

T h e r e would also seem to be the <sup>anger</sup> ~~damage~~ as happened in my case of acute mania, and whether this might not lead to puerperal mania has also to be borne in mind. In my case <sup>such</sup> ~~that~~ was not the case.

W i t h venæsection and careful injection of saline no such danger would seem to exist, and for my part I shall use them according to the method employed so successfully by Jardine.

I n compiling this Thesis I am specially indebted to the following articles :-

(1) Sikes articles on Practitioner

(April and May 1905)

(2) Berkeley Comyns (Journal of Obstet:

of British Emp:)

*Dec-1903. Jan 1904*

(3) Herman's Article Eclampsia (Clifford

Albutt)

(4) Ords Article (Myxoedema (Clifford Albutt)

(5) — Schafers Physiology (Articles on  
Thyroid and Urine.

(6) — Shaws (Organo - therapy)

*published 1905*

*+ numerous other papers herein quoted.  
especially Nicholson's articles in  
the Linn. Obstet. Transacts.*



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